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D. M. Campbell, Editor **A. T. Kinsley, Associate Editor**

Clipping of the canine teeth in suckling pigs is an important factor in the occurrence of "bull nose" and stomatitis. —Hastings.

An increased ration of salt is beneficial to horses that perspire profusely. The salt ration for work horses should be increased about 50% during the summer months.

An immunity against swine erysipelas has been produced by the use of a vaccine or bacterin prepared by using four parts of a culture of *Erysipelothrix porci* and one part of Lugol's solution.

Hog cholera virus is not difficult to destroy and proper sanitary control measures are of very great value in diminishing the ravages of this disease. Cholera virus usually becomes avirulent when exposed to light and particularly sunlight for ten to fourteen days.

The horse fly, including several species of the Tabanus are of considerable importance in certain sections of the country and unfortunately no successful means of trapping these parasites has been found and repellants have failed. The control of these parasites is most effectually accomplished by destroying their breeding grounds by drainage.

The ordinary cooking of foods for either human or animal consumption diminishes the mineral content. Steam cooking removes less of the minerals than other forms and is therefore advantageous.

Is lousiness a sequel or the cause of unthriftiness in swine? Lice can be exterminated or reduced to a minimum by periodical dipping. Hog wallows are a nuisance and should be eliminated.

The rate of excretion of arsenical preparations is an important factor not only in gauging toxicity but also parasiticidal value. Pentavalent arsenicals are more penetrating for cell protoplasm than trivalent arsenicals.

The dairy cow is the most economical animal for the conversion of farm crops into human food; swine are the most efficient animals in the production of meat food products from raw material; and the hen turns raw products into finished products most rapidly.

An infectious pneumonia of sheep in portions of Japan has been found to be due to infection with *B. pyogenes, B. ovisepticus* and *M. catarrhalis.* A mixed vaccine prepared from the three types of microorganisms diminished the losses very materially.

The annual milk production of average dairy cows has been increased 1333 pounds in the last eight years. The average milk production per cow is now 4500 pounds.

Under ground water, wells and springs may be polluted by excreta deposited at variable distances from the supply. It has been demonstrated that well water may be polluted at a distance of 200 feet in a soil that is rather porous.

Bile pigments are largely derived from hemoglobin and it was formerly considered that jaundice and bilirubinuria were primary indications of defective hepatic function but some investigators are now of the opinion that bilirubin may be formed in other than hepatic tissue as a result of hemolysis.

Whole milk of bovine origin, particularly when rich in butter fat, should not be used as an entire diet for orphan pigs. Orphan pigs can be fed and developed on modified milk prepared as follows: Cows milk one pint, lime water one pint, separated cream one-fourth pint, and milk sugar two ounces.

The formation of urinary calculi (renal, cystic and urethal) is dependent upon a foreign body upon which the mineral accretion occurs. The "hardness" of water is not a factor in the occurrence of urinary concretions. On the other hand it has been shown that experimental animals thrive better on water containing some calcium than on distilled water.

Immunity produced by the use of abortion bacterin or living culture vaccine is of relatively short duration. It is possible that a higher degree of resistance may be established by resorting to intravenous injection of the antigen (bacterin or vaccine). Some investigators are of the opinion and have apparently demonstrated that one large injection of bacterin produces a greater resistance than a series of smaller doses.

The larvae of the sheep stomach worm is quite resistant to external influence. Thus these larvae will live in water for five months, at freezing temperature for six months, in fecal matter and in soil for eight months. It is probable that they will survive on pasture land during an ordinary winter in the northern states.

Although vaccination with the single injection rabies vaccine, apparently has reduced materially the incidence of canine rabies, the disease will probably not be eradicated until ownerless dogs are destroyed and all other dogs are properly confined and an effective quarantine is instituted on all imported dogs.

Cultures of *B. abortus* subjected to a temperature of 140 degrees F. for twenty minutes are no longer viable, and it is probable that these organisms are destroyed in milk that is heated to the same temperature for the same length of time and therefore that there is no danger of infection of man with this organism from milk that is properly pasteurized.

Cattle receipts at the seven leading markets during May 1928 were 14% less than for May, 1927, and 12% less than the five-year average for May. This is accepted by investigators as evidence of an impending cattle shortage.

A fatal case of actinomycosis occurred in a thirty-month-old negro girl in Chicago. According to the history of this case, the child had been in the city its entire lifetime and was therefore not exposed to the usual sources of infection with the actinomyces.

A foot maggot of cattle is more or less prevalent in the Philippine Islands. This parasite (Booponus intonsus) is parasitic only during the larval stage. The tissues adjacent to the hoof are invaded by these parasites and marked lameness results. The life cycle of the parasite is probably very similar to other parasites of the same group.

Infestation of a two-year-old Porto Rican boy with Dipylidium caninum, a common tape worm of the dog, was recently reported by Dr. Belaval.

It has been said that the greatest undeveloped resource is the odd looking spherical knob attached to the upper end of the spinal column of the genus homo.

Calves do not constitute a serious problem in the control of abortion disease because they are rarely, if ever, permanently infected until they become sexually mature.

Blackhead in chicks is sufficiently prevalent in the Mississippi Valley states to require those veterinarians interested in poultry practice to give consideration to methods of control as this disease in chicks, probably identical to blackhead in turkeys, does not respond readily to treatment.

A parathyroid extract has been found of value experimentally in increasing the calcium phosphate content of the blood and may therefore be of value when administered in cases of fractures. This line of treatment is at least worthy of a trial in fracture cases and also rachitic conditions.—*Science.*

The larval form of the *Esophagostomum columbianum* is passed wholly in the intestine of the host. They do not migrate into other organs or tissues. They normally become encysted in the mucosa of the larger intestines, however they may invade the mucose of the latter portion of the small intestine.

Potato poisoning is a relatively common condition in animals and man. Poisoning is due to solanin, an alkaloid that is apparently confined to the green portion of the tubers or plant. Poisoning is prone to occur after consuming tubers that have been exposed to sunlight and are distinctly green in color.—*Science.*

The attitude of a veterinarian to the public makes for his success or failure.

Excessive salivation in dogs as a result of medication with oil of chenopodium can be reduced to a minimum by administering the medicament in capsules.

An effective treatment for heaves consists of the administration by mouth of one ounce of kerosene three times daily. —Logan Allen.

Naphthalene is of value in scours particularly in those cases in which the fecal matter has an offensive odor.—Caldwell.

At least three square feet of floor space should be provided per hen and four square feet is better for the larger breeds.

A type of pernicious anemia due to a filtrable virus has been observed in sheep and goats in Algeria. The principal lesions are found in the suprarenal capsules.

Does artificial fertilizer exert the same influence as natural manure on the production and storage of vitamines in food plants is a question that has not yet been answered.

It has been shown that the virulence of a culture of B. Aviseptica will be maintained for twelve months in a sealed culture tube.

Loss of flesh, anemic appearance of comb and wattles and appetite fairly good, are manifestations of ascarids in fowls.

Observations in United States, Sweden and Denmark tend to show that milk should be enrolled in the list of possible sources of the virus of poliomyelitis. Pasteurization affords an effective means of preventing infection from this source.

Footrot in Sheep

By E. T. BAKER, Moscow, Idaho

FOOTROT in sheep assumes two general types:

1. The common footrot or canker, caused principally by the *Staphylococcus albus*.

2. Lip-and-leg ulceration, of which the *Actinomyces necrophorous* is the causative factor.

Some outbreaks may be a mixture of the two, and, when accompanied by a severe general toxemia, may cause considerable loss.

However, the lip-and-leg ulceration is rather rare, and the following remarks will be devoted to the first or more common type.

It affects all breeds of sheep, and may occur at any time, but the winter and early spring seems to be the worst season. From letters and telegrams received from many practitioners from various parts of the country, it seems to be on the rapid increase, and it behooves the veterinarian to "wise up" on this particular malady.

It is one of the most aggravating ailments to get mixed up with, and, as we have treated a good many thousand the past fifteen years, we speak from painful experience.

From the sheep man's standpoint, in the past, he has been put to a lot of unnecessary expense, trouble and publicity by confusing the two types. This has resulted in him being quarantined, and if he happened to be of a different political faith than the powers that be, he was given plenty newspaper space. This killed his chances for selling breeding stock, perfectly healthy, and tied up his operations for some time. The whole affair left him a bitter enemy to sensible quarantine measures.

On the other hand, some sheep owners of a naturally lawless disposition, have scattered serious infections and would be a menace to other wool growers, were it not for strict regulations.

Between these two extremes are found the rank and file of sheep owners who are willing to cooperate in every way, provided they are handled with common sense and justice.

The private practitioner, too, has had his grievances which everyone in practice is aware of, so there is no use discussing this phase of the question.

We mention these to emphasize the importance of an accurate diagnosis, so that the client's interest will be protected as well as that of the public. Tactful handling on the part of the veterinarian will cause the sheep man to have confidence in him, and thus go to him before the infection has had a chance to become widespread.

Symptoms

The usual symptoms are as self evident as azoturia or milk fever. At first, there are a few lame sheep, and these hobble around for a while and gradually get better unless the infection is very extensive and severe. There is an absence of lesions in the genital regions or the mouth. Sometimes a ewe with a cut on the udder may lie down in an infected place and get an abscess, but the principal lesions are found in the feet.

After the first ones have recovered, others become lame, and after the owner has exhausted his efforts, he calls in the local veterinarian. The latter is expected to wave a magic wand, look wise, give the owner some "strong dope" and all will be well.

After a second experience of this kind, the veterinarian will be about as shy in giving a favorable prognosis as he would in telling an old maid he could cure her flea hound of chorea in ten days.

This "yumping in" where angels fear to tread, has been brought about through

the absence of warning literature. The various textbooks have passed up the disease with a very few words, and these placed in such a way that they give no real, practical information.

The real difficulty encountered is controlling the type of infection, and no two outbreaks will act the same. In one case, the disease breaks out with severe virulency and decreases with time; in another, the start may be very benign, but later is hard to handle. Just as the veterinarian reaches for the laurel wreath of victory, and begins figuring up how much will be the bill, the owner calls up and informs him fifty more ewes are "lamer n' hell!"

As Prof. Buchanan says in his work on Bacteriology, page 231: "Possibly anaphylaxis will explain some of the reactions secured but there seems to be some poisonous property, possibly an endotoxin, which is not at present understood."

In the language of the wide, open spaces, the Professor said a mouthful. In fact, we agree perfectly on the subject. We might go further and say that footrot is about as uncertain as women, baseball or politics!

Therefore, one cannot even hazard a guess on how long it will take until the disease has been thoroughly eradicated. In our experience it takes from three months to as many years.

Course of Disease

The course and virulency of footrot is very atypical, and varies so much that hard and fast rules cannot be given. Usually, the larger the band the longer the infection persists.

Several hundred may be affected in a band of 2,000.

The chances are in a bunch of sheep, large or small, one or more is very susceptible to the pus organism. These animals act as incubators or carriers. Following a wet spell of weather, other sheep bruise their feet, stand in mud or filth, and thus soften the integument around the hoof. Suppuration then takes place, and the pus germs proceed to make themselves at home.

If the interdigital gland between the cleft of the hoof becomes clogged with a thick, cheesy secretion, the patient will show extreme lameness and pain.

If the sheep is strong and rugged, it may hop around for a few days, then gradually work out of it, and nothing more is thought about it. Later, when several hundred get lame, the owners sit up and take notice.

One of the herders, who knows more about medicine than old Hippocrates himself, diagnoses the trouble as "rot," but tells the owner not to worry, as he has cured millions of cases, so he is given permission to hop to it.

Hold a sheep gently but firmly.

The whole outfit of men now start in trimming hoofs, and they cut and slash until they can hardly eat. The odor, to use a description from a well known perfume ad, is "distinctive." It is not conducive to a big appetite, but the operators manage to get along with the aid of much "sheep herder's delight," which is usually available. As God fed Elijah with the aid of ravens, the average sheep herder manages to get along very well with little food but much "mountain dew."

They fuss around with the trimming, and then a strong solution of blue vitriol is mixed up, and the animals driven through. This sometimes forms a hard crust, and, in many cases, acts as a friendly shelter for the wily staphylococcus.

After several weeks of cutting and dipping and cussing until about half the band are affected, a veterinarian is called in.

Our expert may labor under the delusion the pus germ is not difficult to exterminate. Sunlight, mild antiseptics or benevolent germicides are all that is needed. This may all be. In the laboratory the pus germ turns up its toes with alacrity and dispatch, but when it homesteads in a sheep's foot it acts otherwise. Anyone who has ever treated a deep-seated abscess, fistula, quittor, poll evil, thrush or even grease-heel knows how simple it is!

Before wringing the germ's neck it is necessary to find it. As the old fellow said in explaining how to be a successful huckleberry picker: "You gotta git whar th' berries is."

The writer has taken many sheep and worked out the following experiment: The abscess would usually be found just above the coronary band, in the shape of a flat swelling, very painful. The skin would be disinfected well with spirits of camphor, and a small incision made. White pus would appear, and a smear would be made of as pure culture as possible for bacteriological examination. The abscess then would be laid wide open,

Convenient method for medicating the feet in a small flock. Place a wisp of wool in the interdigital space.

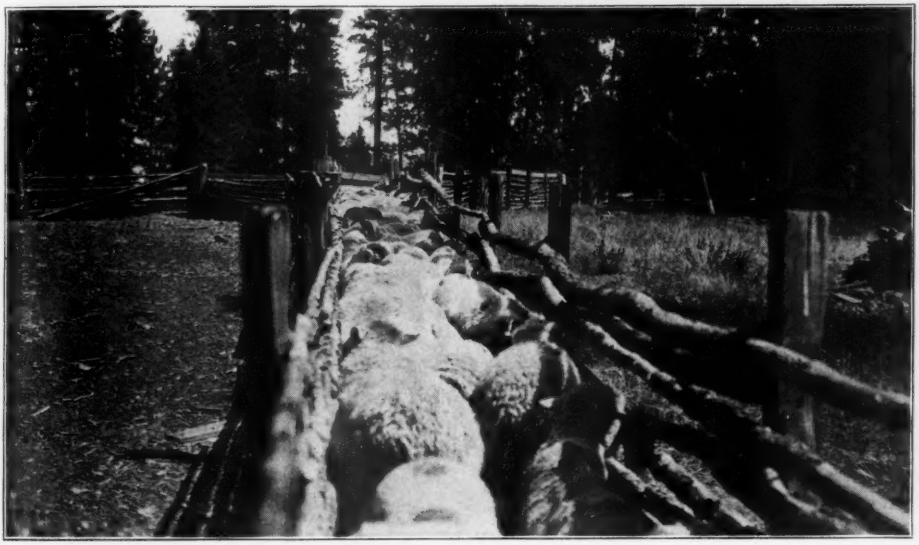
and all the secretions swabbed out with cotton, and equal parts tincture of iodin and ether applied. All the rest of the foot seemed normal. To make sure of as careful a disinfection as possible, all the feet would be dipped in some good germicide, using different kinds. The animal would be marked and turned loose.

A week or so later, this same sheep would be examined, and the chances are another abscess would be found in a different place or in another foot. Paring the hoof away we would find a grayish mass of decayed tissue, with the typical aroma of foot rot, which is not exactly like the odor of an Early Richmond rose. Whether this lesion was present when we lanced the first abscess, of course, is not known, but we can say there were no clinical manifestations.

This second abscess would be rudely laid bare with a sharp curette, scraped well, and pure balsam of Peru with a 5% silver nitrate added, anointed all over the foot. This is one of the most healing mixtures the writer is aware of, and were it not for the expense, would make an ideal treatment.

Our patient would now be examined for a third time, and in about eight times out of ten would be entirely well. The two cases were what caused the trouble to hang on, and make one wish he was an evangelist with a one-armed partner to take up the collection.

In the west, the practitioner is confronted with dealing in whole sale lots. We have treated as many as 800 head out of a band of 2,000, and one outfit with over 14,000 head had some two thousand cases scattered over a period of several years. Not many at a time, but just enough to worry them. Farm flocks of 30 to 250 often have several dozen head limping around, and feed lots are a common source of infection.

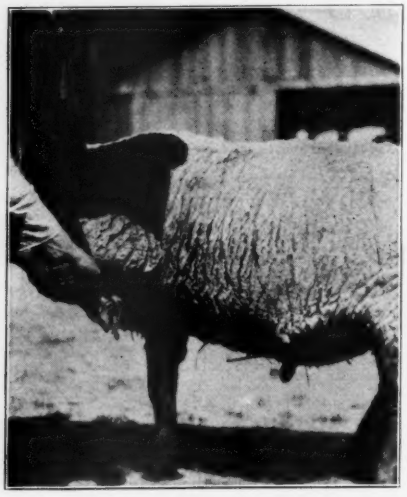


Large bands are run through a dipping vat.

We might say that our first experience occurred some years ago, and started off with enough grief to drive a man to drink. About the middle of September we received a call to go about sixty miles in the mountains to see a sheep man who was having trouble. The fall rains had started early that year, and our Buick chugged through mud up to the running boards. We found a bedraggled looking

bunch of sheep cooped up in a small corral, and it was a sight to make an angel weep. Out of some 2,200 head nearly half were lame, and some were so bad they could only navigate on their knees.

The history had been rather desultory. All the winter before they had had a few lame ones, but these recovered without any treatment, and all summer in the mountains, perhaps a dozen or so had



Trim the hoof thoroughly.

limped. Starting to trail to the home ranch, they struck some rough roads through an old mining country, and in a few days several hundred were unable to go on.

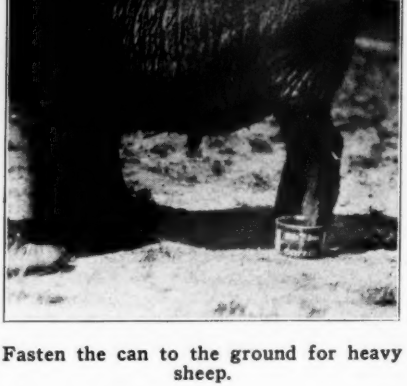
The herders had called up the owners and they in turn had called me, telling me what to expect and be prepared for. I might say that I read more books in about two hours than any freshman preparing for his first exam. On the subject of footrot in sheep my mind was a virgin field. However, I outlined a treatment and packed in the various drugs I figured I would need.

We found the typical symptoms of common footrot or canker. The absence of lesions in the genital tract or mouth eliminated lip-and-leg ulceration, for which we breathed a sigh of relief.

About this time the rain began to pour harder than ever, and we had to put up an old lean-to, covered with fir branches, which made a temporary field hospital. An old Scotch sheep man had told us some time before not to be afraid to trim the hoof, and trim we did.

We then made up a solution of arsenic by boiling six pounds of arsenous acid and nine pounds potassium bicarbonate in twenty gallons of water, for nearly an hour. We used five gallon cans and guessed the amounts of the drugs. Half a dozen plank were hauled in about thirty miles, and a trough or vat made. We also ordered a barrel of lime and slaked this in the rain.

We then added enough water to the arsenic solution to fill the vat up about four inches deep, and drove the lame ones through. The next day we fixed up a narrow runway in which we put the



Fasten the can to the ground for heavy sheep.

lime and ran them through this. We had a gallon of creolin along with us, and using a big can of axle-grease and some fryings and soda, we mixed up enough ointment to apply to the worst ones.

In a few days the animals showed a great improvement, and we got a permit to ship them back to the home ranch. It took all winter to get the outbreak com-

pletely eradicated, but those sheepmen have been among my best clients ever since.

In other words, we got them out of a jack-pot by using the means at hand, and they appreciated it. From what I have been told by men who were over there, sheep practice on the range must resemble battle field surgery—one works with what he has, and makes the best of it.

Treatment

First of all, make your diagnosis, using the aid of a bacteriological examination if possible.

Second, thoroughly trim the hoof.

Third, medical treatment is now in order. The writer has used aqueous and glycerin solutions, oils, ointments, powders, balsams and caustics. If practical, one could use a tight-fitting container and gas the foot with pure formalin.

However, one very soon discovers what will clear up one outbreak may prove worthless in another. The kind of medicine used seems not so important as to be lucky enough to come in contact with all the infection. We mention the word "luck" because that covers a lot of quick cures. Just because you happen to clean up your first outbreak, don't get cocky and severely stretch your hat-band!

In a small bunch, we think balsam of Peru and a one to 5% silver nitrate added, about as good as anything. The mixture should be warmed, and three or four inches poured in a can and the foot dipped in it every day for a week at least. This is expensive, but very healing.

For larger numbers, we can mix half an ounce pyoktanin blue in half a gallon liq. cresolis comp. and add two gallons each of coal oil and light mineral oil. Cajeput and eucalyptus can be added. This is excellent for maggots, also.

As a dip in which to run the affected animals, or the whole band for that matter, any antiseptic can be used. Blue vitriol, potassium permanganate, arsenic, creolin, lime and sulphur, chlorines, etc., are all about the same. However, unless it has a strong odor and "smells" like what the owner thinks should be a disinfectant, he is not satisfied. Personally, we like some of the aromatic disinfectants put out by the various supply houses. It is a good idea to keep the nature of your curative agents under your hat, for one sheep man will tell another, and in the future all the veterinarian will get is what the little boy shot at and the owner will bungle the job somewhere along the line.

In making a vat, multiply in inches the depth, width and length; divide by 1728, and multiply by 7½, which gives the amount in gallons. Thus a vat twenty feet long, eighteen inches wide and with the solution four inches deep, will require from 75 to 80 gallons.

Abortion may occur with fatal result.

After the infection has been killed, any kind of healing ointment will assist in the curative measures, and more quickly restore the animal to normal. In doing this, we impress on the owner that the disease is not a simple matter, and he will call on help a little quicker the next time.

A good ointment for general use is made up as follows: Melt fifteen pounds yellow petrolatum, ten pounds lard or mutton tallow, one pound beeswax, sepa-

rately. Mix together in a large pan, and then add, stirring vigorously, one pound oil of cade, one quart lysol, eight ounces carbolic acid, one quart carbo-campho, and lastly, ten pounds of finely sifted sulphur.

For use, warm this slightly, put about four inches in a can, and dip foot in it. Use at least every other day for a week or so.

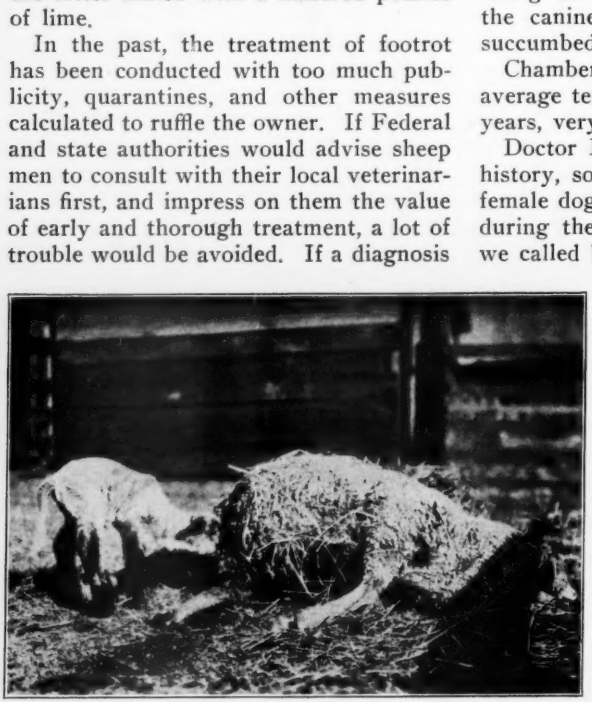
This ointment is very good to use after lancing "blue bag" or gangrenous mammitis in the ewe, and any other kind of sores or abscesses.

However, any favorite healing oil, salve, powder or solution will do the work. Plenty salt with one pound potassium iodid added to the ton is also indicated for a tonic.

If at all possible, change the animals to new quarters, and sprinkle the ground with lime and sulphur—eight pounds of the latter mixed with a hundred pounds of lime.

In the past, the treatment of footrot has been conducted with too much publicity, quarantines, and other measures calculated to ruffle the owner. If Federal and state authorities would advise sheep men to consult with their local veterinarians first, and impress on them the value of early and thorough treatment, a lot of trouble would be avoided. If a diagnosis shows the presence of necrophorous, then a quarantine should be placed, and appropriate treatment started.

In conclusion we want to add that footrot, pustular dermatitis, canker, panaritium, or whatever you want to call it, is one of the most difficult diseases we know of to treat and clean up satisfactorily. Use different lines of treatment in your practice and keep this strictly to yourself or colleagues, and it will return you a fair income.



In severe outbreaks death may occur from generalized septicemia.

DOG ATTAINS AN UNUSUAL AGE

The readers of VETERINARY MEDICINE may be interested in the following item appearing in the Ames (Iowa) Tribune of September 4th:

"The mongrel dog which has been the faithful friend and guardian of the L. H. Pammel family for 20 years is dead today at the advanced age of 22. After living far beyond the allotted span of the canine race, the old fellow finally succumbed to the heavy burden of years."

Chambers Encyclopedia states that the average term of life of a dog is 10 to 12 years, very rarely more than 20 years.

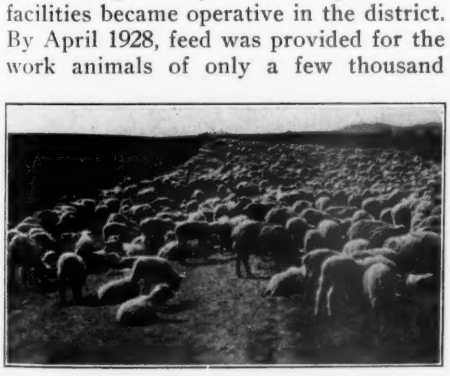
Doctor Pammel says of his dog: The history, so far as we know, is that this female dog came to us twenty years ago during the month of June. "Chips," as we called her, had had a litter of young some 6 months previously. The dog, a mongrel with some Spitz blood, was spayed in the Veterinary Department, Iowa State College, a few months after coming to us. The dog was an unusually intelligent one. She knew every one of the family. Even though we were gone for three months, the dog always recognized the members of the household. In late years she lost the keen sense of hearing. In all of this time she was sick only once. She was a faithful companion to children and the household.

Red Cross Charged With Care of Live Stock in Flood Area

WHILE Red Cross activity is generally devoted to the care of human beings, looking after the welfare of the people will, in many cases, involve also looking after the welfare of animals which these people own. The American Red Cross went into the business of caring for animals on a large scale during and after the floods in the Mississippi Valley in 1927.

With the sudden sweep of the disaster, some 900,000 persons found themselves the victims of the greatest catastrophe which has struck that section of the United States in years. The Red Cross, as in other disasters, was on the job immediately. But, the district being agricultural, it was realized that in order to bring about proper rehabilitation when the waters receded, it would be necessary to make provisions for the live stock of the thousands of farms. Because of this, the Red Cross immediately took upon itself not only the task of caring for some 600,000 persons, but also that of caring for some 200,000 horses, mules and cattle.

During the emergency period of the flood, the Red Cross fed more than this number of live stock and this feeding continued throughout the remainder of 1927 and well into 1928, the number of live stock gradually diminishing as credit facilities became operative in the district. By April 1928, feed was provided for the work animals of only a few thousand owners who were busy with their spring plowing and who were, as yet, unable to buy their own feed.



In caring for these animals the Red Cross expended the gigantic sum of $2,347,600. Thousands of bales of hay and large quantities of other feed were shipped into the affected district in order to accomplish the task of feeding the animals. When the flood waters had receded, the organization spent an additional sum of $310,663 in restocking farms with horses, mules, cattle and poultry so that the owners would be in a position to carry on their work without too great a handicap.

Rebuilding and repairing of houses and barns in the flood area necessitated an expenditure by the Red Cross of more than $1,800,000. As a result of this activity, many of the farms are operating upon an equal, if not better basis than before the flood. And another important factor is the betterment of sanitary conditions which have been brought about by the reconstruction work of the Red Cross following the disaster.

Total expenditures of the Red Cross in the Mississippi flood disaster, according to tabulations made in June of this year, aggregated $17,473.46. While the expenses of such an extraordinary character cannot be borne by the organization without appealing to the American public, the Red Cross makes it a policy, in carrying on its every-day work as well as much of its special work, never to appeal to the public for endorsement of its program except upon the occasion of its annual roll call. The Twelfth Annual Roll Call of the American Red Cross this year will be held beginning Armistice Day, November 11, and continuing through Thanksgiving Day, November 29. The goal set is 5,000,000 members.

Infectious Perulent Entero-Proventriculitis of Fowls

By B. F. KAUPP, Raleigh, North Carolina,
Avian Pathologist, North Carolina State College

Introduction

THE filterable viruses as a cause of diseases in poultry have received slight recognition up to the present time, but it appears within the bounds of possibility that the future may show important disease outbreaks in flocks which run their course without diagnosis are the result of infection with an ultra-microscopic organism.

On October 20th, 1926, there was referred to the Poultry Disease Laboratories of the North Carolina State College, an outbreak of a disease in a flock in New Hampshire which, from the description, appeared to be a malady not yet recorded in the literature of poultry diseases, and which was highly contagious and of sufficient virulence to produce a high death rate.

The owner reported that the foundation stock were S. C. Rhode Island Reds and were secured from a reliable source and the flock health, prior to the outbreak mentioned above, had been exceedingly good, the fertility, hatchability and livability in the chicks from this flock being extremely high. On the sixth year, pullets from this flock on coming into lay, showed an inclination to sneeze and shake their heads violently, which was a constant symptom as the disease progressed. There was no indication of true "colds" or the allied respiratory diseases. External abscesses as shown in Fig. 1 were next observed appearing at various sites—the eye lids, below the ear, the foot, and inside the vent. Postmortem examination by the owner showed congestion of the internal organs and a great many internal abscesses. Emaciation usually preceded death. In this flock of pullets the mortality due to the disease was about 33 1/3%.

On March 12, 1927, the owner again communicated with the laboratory stating that another pen of pullets, seven months younger than the above-mentioned pen, were coming down with the disease. The symptoms were similar to those exhibited by the first pen, with the exception that paralysis in the legs or wings occasionally appeared. This stage is shown in Fig. 2 in which the bird is unable to rise and shows the typical swollen eye ring. Mention was made that in the first pen, the males showed high susceptibility. This was not apparent in the second pen in which the disease appeared.

Pullets from five months to one year of age are the most susceptible. The disease usually appears when the birds come into lay. There seems to be no sudden onset with large numbers coming down at once, but the disease presents a slow but sure progress with an ultimate mortality of about fifty per cent.

Twelve field cases were received at the laboratory including those shown in Figs. 1 and 2.

Case Reports

Autopsy. Case No. 1. S. C. Rhode Island Red pullets. External appearance normal. The liver enlarged and friable; the spleen and heart normal; kidneys and lungs normal, and purulent inflammation of the small intestine. The follicles of the small intestine engorged with pus, when the wall was pressed on pus oozed out at each glandular stomata. Cultures in all these autopsies were made from the brain, spinal cord, heart, liver, spleen, kidneys, lungs and bone marrow.

Inoculations were made on blood serum, neutral bouillon, neutral glycerin agar, and nutrient infusion agar, pH 6.4 and incubated

48 hours at 37.5 C. Cultures from bird one were negative. Sections of the internal organs, proventriculus, brain and spinal cord were emulsified and incubated 48 hours in nutrient bouillon, and Berkfield filtrate inoculated into a S. C. White Leghorn cockerel to test for filterable virus.

Autopsy. Case No. 2. A S. C. Rhode Island Red pullet. Highly emaciated. The liver enlarged and friable. The remainder of the internal organs normal in appearance except for purulent glandulitis of the proventriculus and purulent inflammation of the small intestine. Three sacs of pus about 1 cm. were attached to the mesentary. Cultures are negative and a Berkfield filtrate of a 24-hour bouillon emulsion of the glandular organs, brain and spinal cord was inoculated into a S. C. White Leghorn cockerel to test for a filterable virus. The autopsies of the 10 other cases sent to the laboratory were similar to one or the other of those recorded above.

Clinical Studies of Experimental Birds

The respiration studies indicate that the breathing is labored although to what extent it cannot be ascertained on account of the lack of pre-infection studies on the bird.

There was not sufficient change in temperature to warrant the belief that, in these cases, the disease affects the heat regulating centers.

The blood studies showed a decrease in hemoglobin, although both the leukocytes and erythrocytes were above normal, the increase was proportionate and cannot be regarded as conclusive, as occasionally an individual bird maintains an exceptionally high count while apparently in normal health. There is apparently a mild polynucleosis and an increase above normal in the number of mononuclears.

There is apparently a collapse of the nervous system resulting in complete paralysis. The watery discharge which seems to be constant in the last stages of the disease is evidently the result of muco-enteritis observed especially in the small intestine.

Microscopic Pathology

Kidneys—There is a cloudy swelling and active and passive congestion; areas of focal necrosis are apparent. The cells of the convoluted tubules are swollen and in places the lumen is obliterated, and the cells at their bases have left the wall. Areas show cloudy swelling and obliteration of the lumen of the tubule.

Proventriculus—Congestion and suppuration are apparent as evidenced by the ascini being filled with pus. The surface tubular glands are in a state of cloudy swelling and necrosis, with pus formation.

Small Intestine—In gross this is covered with a thick purulent material. Microscopically there is a cloudy swelling of the basal cells of the mucous membrane with necrosis of areas of superficial cells.

Heart—Congestion is present, the muscle fibers have not lost their cross striae.

Lungs—Congestion may be present.

Fig. 1. Abscess of the face due to Infectious Purulent Entero-proventriculitis.

Liver—Active congestion is present. cloudy swelling with fatty degeneration is noted. The active congestion is especially markedly pericapsular.

Artificial Infection Studies

Lack of success in growing of a microscopic organism from various organs on

media suggested an ultramicroscopic organism as the causative agent of this disease. Accordingly the Berkfield filtrate from birds 1 and 2, as described in the autopsy records of these birds were given orally in 2 cc quantities to S. C. White Leghorn cockerels "A" and "B" respectively.

At the end of six weeks, both birds were gassed for autopsy, and revealed an inflammation with pus formation of the intestine and proventriculus.

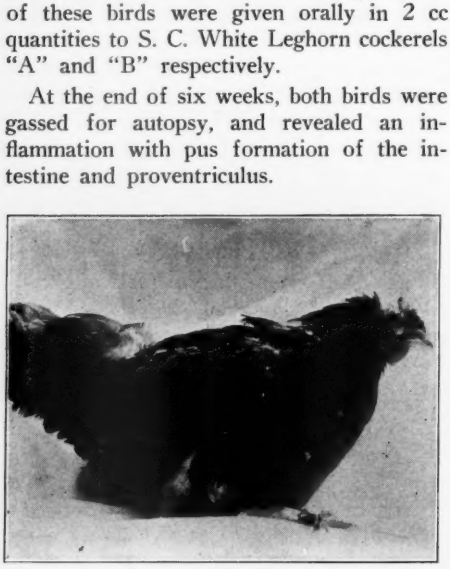


Fig. 2. Clinical symptoms of the advanced stages of Infectious Purulent Entero-proventriculitis. Unable to stand. Note swollen eyering.

A Buff Orpington pullet was placed in contact with case No. 10 for a period of seven days, the birds occupying the same cage, having a common drinking and feed cup. The contact bird died at the end of thirty-seven days.

It is evident that the disease is quickly contracted as the birds show well defined symptoms on the second day of contact with the clinical case. The outstanding initial symptom was an evident depression and extreme pruritis, which seems inevitable with this disease. These symptoms continued becoming more pronounced until the fourteenth day of contact when the watery discharge, which is constant with the naturally occurring cases is observed, and when the initial symptoms of leg weakness appear. There is evidently an acute nervous affection as the bird shakes its head violently at periodic intervals. The leg weakness became more pronounced until, on the 27th day the bird lost completely the use of its limbs. On the 30th day complete paralysis had set in, the bird having the use of only the head parts.

These were only a few of the experiments and space will not allow of giving more details. However before passing from this phase I should state that subcutaneous inoculations of the filtrate were also made with the same results.

Since these studies were made I have come across the disease in S. C. White Leghorn chicks of about one pound weight. The first thing on postmortem that attracts one's attention is the ballooned proventriculus which has a thickened wall. Another case was sent to the laboratory from North Carolina. It is quite possible that this disease is quite widespread but that it has heretofore been overlooked.

Not being able to find a description of the disease in literature and believing that it is a new disease we have proposed for it the name infectious purulent entero-proventriculitis. This name when analyzed tells just what the disease is.

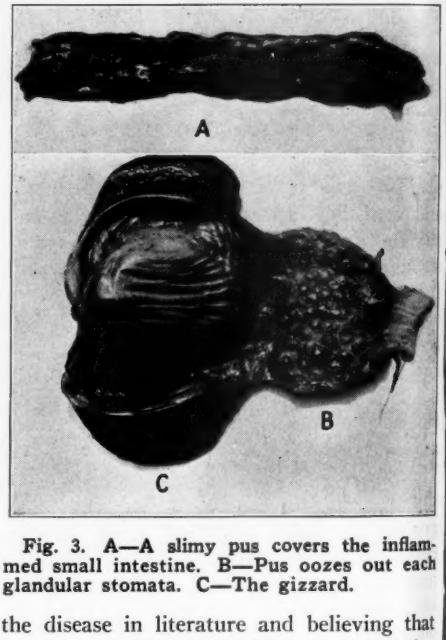


Fig. 3. A—A slimy pus covers the inflammed small intestine. B—Pus oozes out each glandular stomata. C—The gizzard.

BOOK REVIEW

Poisonous Plants

I have just had the pleasure of looking over a book by W. Dallimore, edited by Dr. A. W. Hill, F.R.S. on Poisonous Plants, Deadly, Dangerous, and Suspect, published by Frederick Etchells & Hugh Macdonald, 10 Kensington Place, London, 1927.

It is a superbly printed volume of quarto size with 85 pages and 20 figures finely executed on woodcuts. The Poisonous plants are divided into:

1. Plants Deadly (deadly nightshade, henbane, cuckoo-pint, hemlock, water hemlock, and monk's hood).
2. Plants Dangerous (foxglove, meadow saffron, spurge laurel, and thorn apple, caper spurge, herb Paris, pasque flower, bear's-foot, black bryony, and yew).
3. Plants Suspect (horned poppy, common buckthorn, common buttercup, and bitter-sweet).

This interesting statement is made in the Introduction: "The plants thus chosen form a community of elegant malefactors, inhabiting a variety of situations from churchyards to the sea-shore. At one end of the scale of interest are the two umbelliferous plants, both inhabitants of damp meadows and stream banks, and members of a generally dowdy family. At the other end is the strange thorn apple, with its twirling white funnels and prickly fruit, cousin to tropical plants even more exotic and terrifying. The bear's foot, or stinking hellebore, is a peculiarly handsome plant in leaf and flower, and is, with the spurge laurel, the earliest to bloom: these two, and our common foxglove and herb paris, form the woodland group. The yellow horned poppy lights up the sea shingles; taxus stands gloomily in burial grounds; the pacque flower is Spring's forerunner on the chalk downs; while the deadly nightshade, pre-eminently in appearance the king of poisoners, is found in copses and on waste ground.

Our poisonous plants are not all indigenous; some are naturalized, some merely escape from gardens. The thorn apple is a native of North America, and will appear suddenly, growing in unexpected places. The seeds may probably be imported in sacks or packing. The caper spurge is another naturalized flower common as a weed in gardens. The Romans are said to have been responsible for the introduction of the henbane into England; but we can forgive them this act, if Farrer is correct in saying that the pasque flower also accompanied them, lured to their distant colony by the superior quality of the mortar rubble. The monk's hood, though common in nearly every garden, is rarely found wild in England."

It is a well known fact that many of these plants at an earlier date were used for medicinal purposes. Some of them were used to produce poison for the ancients. The author has made an error in the origin of the jimson weed. It is a native of India rather than of North America. It is true that we have species of thorn apples in the southwestern part of the United States, but they have no close affinity to the Asiatic thorn apple or jimson weed.

Throughout from the printer's standpoint, this is a splendid volume, but hardly will be generally distributed. The number of copies issued was 350. As a work of art, it is splendid.—L. H. Pammel.

COWBANE IN IOWA

A correspondent writes:

I am sending you a sample of a weed which is growing around here, (North English, Iowa), in small patches on wet ground. I am afraid it is poison hemlock.

I shall appreciate any information concerning this plant that you give me.

This plant proves to be cowbane, or musquash root, an Indian name, or frequently but incorrectly called "wild parsnip," *Cicuta maculata*. The plant has white flowers, resembling a carrot, fleshy fascicled roots and compound divided leaves. Roots and plants have a strong pungent odor. The plant grows in low places. *This is the most poisonous plant in North America.* The poisoning is due to a toxin. A small portion is sufficient to cause fatal poisoning. It causes convulsions.

Ames, Iowa. L. H. Pammel.

Specific Pulmonary Edema of Cattle

A Bacterial Infection Due to Unnamed Diphtheroids or Corynebacteria

By J. D. RAY, Kansas City, Missouri,
Laboratory Director, Kinsley Laboratories, Inc.

IN THE routine of consultation and laboratory diagnosis, during the past year, some interesting and baffling conditions have been encountered in bovines. It has been apparent that lesions in specimens from recognized diseases were becoming more or less atypical. Too many specimens gave negative cultures when they were made from material showing marked lesions. After variations in laboratory technic and the careful observation of lesions and cultures, we have been able to associate certain of these strange lesions with a definite type of bacterial infection.

The organisms encountered in the cases in question belong to what we believe to be the old diphtheroid group classed now as the corynebacterium.

Examination of the literature available does not describe conditions as we have found them in domestic animals as being caused by such organisms. Many cases have been observed where only these organisms were found along with what appears to be pathognomonic lesions, hence we believe that we are dealing with an infection that has not been fully described.

These laboratory findings are of particular interest because of the close similarity of some of the findings in the recent publication of McBryde, Niles & Moskey.—"Investigations on the transmission and etiology of Hog Flu" (Journal of the A. V. M. A., July, 1928).

The condition as it affects cattle will be considered primarily at this time, although a similar condition has been observed many times in swine and sheep.

The following general description is offered, with the hope that some light will be thrown on a few of the problems that have been baffling many laboratory men and practitioners throughout the country.

Pulmonary edema, as described, is believed to be due to an infection with unidentified diphtheroids or corynebacteria. This group of organisms are very changeable in different environments and under variable cultural conditions, so no definite biochemic description of the organisms isolated will be attempted. They appear to be morphologically typical of this group (pleomorphic, Gram positive, non-sporulating, non-motile, diphtheroid like bacteria).

All classes of cattle become affected, but young stock seems to be most susceptible. Cattle recently exposed through shipping are especially prone to develop the disease. Native stock, however, commonly develop the malady. Calves being raised around large dairies frequently become affected.

Specimens showing specific pulmonary edema have been examined, and characteristic organisms isolated from Colorado and Texas on the west to Pennsylvania on the east.

Clinical Manifestations

The principal symptoms manifested as a result of involvement of the lungs in cattle at its onset consist of a shallow dry cough and dyspnea. There may or may not be an elevation of temperature. Dyspnea is extremely aggravated in affected animals by exercise, thus it is not uncommon for such animals to collapse if forced to exercise. In the subacute and chronic form, particularly in calves, there is evidence of solidification in the lungs, emaciation and later digestive disorders, manifested by diarrhea. Most such cases ultimately die.

Post Mortem Findings

The lesions are primarily confined to the lung and consists of an edema. An af-

fected lung, when observed in the initial stages, has a peculiar grayish color, does not collapse, and has a doughy or rubbery resistance. In fatal cases practically the entire lung is involved. The lung lobules will be well outlined by the edematous fluid that has accumulated in the interlobular spaces. (Emphysema may be more or less marked in the lung tissue, due to the interference of normal respiration.)

If such a lung is sectioned and pressure applied, quantities of edematous fluid can be expressed. Such lungs have been termed "soggy" by some practitioners. In those cases that survive for several days the edematous fluid will gravitate into the dependent portion of the lung. This leaves the dorsal part practically normal and the lower areas solidified.

There may be hemorrhages into the lung and heart coverings or other tissues, apparently due to rupture of capillaries. These hemorrhages are practically the same as those observed in cases of acute fatal hemorrhagic septicemia and hence the confusion in diagnosis.

In subacute cases the pulmonary edema is followed by necrosis in which there may or may not be abscess formation, particularly in the dependent lobes of the lung. Uncomplicated cases never show the usual inflammatory processes, commonly found in other disease processes.

The course of the disease is variable. In some instances the animal apparently has sufficient resistance to overcome the infection and, after having had a hacking cough for several days, apparently entirely recovers. Most cases that show distressed breathing, unless treated, usually die. Even if the animals recover, there is a marked shrinkage during an attack of the disease. In other cases the disease apparently runs a more rapid course. The animals may die, apparently of asphyxia, within two or three days after the onset.

Differential Diagnosis

It is often necessary to make a bacteriologic examination of specimens to learn the exact organisms present.

Many cases showing lesions thought to be atypical of hemorrhagic septicemia have been examined and either no diagnosis could be made from cultures or hemorrhagic septicemia would be found. All known methods of control would not stop the spread of the disease in exposed herds or affected individuals. In such cases both diseases must be recognized.

Cottonseed meal poisoning has been mentioned as a possible condition to be differentiated.

Prevention and Treatment

Pulmonary edema, as described, can best be prevented by following out strict sanitary measures and the use of a properly prepared bacterin.

In treating individual cases showing dyspnea, absolute quiet is essential. This, with proper care and specific bacterin therapy, has been most successful in handling many cases. Treatment is of little avail after necrosis and abscess formation takes place in the affected lung tissues.

A BACTERIN FOR PINK EYE IN CATTLE

I am having quite a lot of trouble with pink eye in cattle. Please advise me what treatment you recommend, and if there is a reliable vaccine for its treatment or prevention.

I have used sterile milk and anti-influenza serum with very good success. I would be glad to find the very best thing to be had.—J. E. Z., Tennessee.

Reply: "Pink Eye" in cattle is apparently associated with *B. pyogenes* infection. In addition, there is usually present staphylococci and sometimes streptococci also. A Keratitis bacterin is obtainable on the market, which has been widely used, particularly by veterinarians in the Western states, both as a preventative and as a treatment of "pink eye." The results of the use of this bacterin so far as we know them have been satisfactory.

Contagious Abortion in Cattle and Its Relation to Undulant Fever in Man*

DOCTOR KING said in part "that of 530 patients whose blood was examined at the Mt. McGregor institution 13% reacted to agglutination test; that an examination of the herd of 150 cows gave 56 reactors, 68 negative, the balance suspicious; that an examination, however, of the entire group showed only 23 animals eliminating *B. abortus* in the milk. The cows were separated according to agglutination test and placed in different sections of the stable, separated only by partitions extending across between the sections. The milk of the reacting animals was pasteurized and used for calf feeding only.

"It was found that one of the cows failed to react to the agglutination test, yet was eliminating the organism through her milk. The same experience had twice been met by other workers to my knowledge."

The speaker stated "that no new reactors had appeared among the inmates of the institution since pasteurization of milk was started; that while they had always produced milk under exceptionally sanitary conditions, more than meeting certified milk requirements, it was evident that reliance on the agglutination test alone to detect infected cows was not sufficient to fully protect the milk supply."

Doctor King stated further "that investigations conducted by himself and other investigators, notably Carpenter and Alice Evans, in various cities throughout the country, had indicated that the number of persons infected with undulant fever was lower where the milk supply was pasteurized than in localities or institutions where it was used in the raw state."

The speaker discussed the difference between *B. abortus* and *B. melitensis,* and pointed out the method of determining the specific organism through guinea pig inoculation. This is done by inoculating the experimental animals with sediment obtained by centrifuging the milk or using cream in which the organisms may be found in large numbers. The guinea pig, if infected, will show enlarged spleen and pin-point lesions if the liver, both these conditions being very constant. It was pointed out that great care was necessary in definitely determining which organsim existed in any specific case because of their similarity. The case was cited of a physician suspected of having tuberculosis running a definite temperature for some time and finally being found to be infected with the abortus organism. Reference was also made to a number of human patients where intermittent and more or less persistent fevers, general lassitude and depression were later traced to the presence of this germ in the system.

Doctor King stated "that in handling the herd at Mt. McGregor, it was the custom to return cows that ceased to react to the negative group. The original group of non-reactors, viz. 68 head, has now been increased to 92 by this transfer."

In reply to a question as to symptoms in man, Doctor King stated that it was often confused with typhoid fever, miliary tuberculosis or rheumatism. He stated that the white blood corpuscles were much diminished, the spleen and lymph nodes were enlarged and the diagnosis was positive only by means of the blood test or blood culture. It was stated that undulant fever was dangerous to laboratory workers and that a considerable number had been accidentally infected, the disease, however, in most cases caused only temporary inconvenience. The organism was not infrequently found in the tonsils. Bone infections were also common in the more persistent cases. One attack apparently does not confer immunity.

* From address at regular quarterly meeting of Hudson Valley Veterinary Medical Society, Kenozia Lake, August 8th, 1928, by Dr. M. J. King, Director, Research Laboratory, Metropolitan Life Insurance Company, Mt. McGregor, New York.

as one worker is now suffering from the third attack which had persisted for some fourteen months.

It was brought out by questions that the cattle at the Metropolitan Farms had been vaccinated some years ago using living organisms; that in the opinion of the speaker this had reduced the percentage of abortions in the herd, but he was unable to give any definite figures to support this conclusion.

The variation in results of tests in different laboratories was discussed and the speaker stated that different antigen, length of shipping, and other factors were in his opinion responsible for these variations. It was explained that much work needed to be done to clear up the questions, method of testing, etc., and cooperation of all concerned was necessary. Many further questions were asked in relation to this disease and contagious abortion in animals.

SHALL THE VETERINARIAN DISPENSE OR PRESCRIBE?

In writing this I may be trampling on someone's feet, especially if they have tender corns. However, it is not my intention to cast reflection on the practicing veterinarian, nor any of the commercial houses, because we don't all look through the same glasses and all see alike.

In discussing this subject, there are at least two cardinal points that should be kept in view, first, what is the best for the client; second, which is the most efficient and economical for the practitioner. This of course is governed in some measure by whether one has a country practice or city practice, or a mixed or small animal practice.

If one has a mixed, country practice, it is best to prescribe as much as possible, especially if one has to wait twelve months for his pay and where the pay is questionable. However, the practitioner should always carry enough medicine along in making his calls for emergency cases.

If one has a small animal practice, and is located in a large city, I think it best to dispense his medicines from his pharmacy, as I have found most people prefer to getting the medicine required right in your own office to taking a prescription to the drug store and having it filled. Besides the small dose required for these small animals allows the practitioner a profit.

This brings us down to the point of what form shall we use our medicine, whether liquid, powder, pill, capsule or tablets.

I have found in my section my clients don't like a liquid medicine, consequently I seldom give out liquid medicine when I can avoid it, although I use considerable liquid medicine in my hospital practice, and if one uses one of these duck bill medicine spoons, it is usually very easy to administer. However, I like a sugar coated tablet best. Very often one has a mean dog that it is quite a job to force medicine down and by splitting a cube of meat and slipping a sugar coated tablet into it, then tie it up, they will eat it, but I have never seen a capsule but what the dog could smell the medicine through it and refuse to eat it.

This brings us down to the veterinary supply house that sells to veterinarians only; I don't buy from the other kind. I like this all right, provided they keep what the veterinarian wants, but as far as I am concerned, there is but very few of them that keeps even as much as 5% of what I want or use in my practice. It may be said that I am hard to please, maybe so, but I certainly know what I want to go down the throat of my patients, and for the past twenty years have been buying most of my tablets, etc., from houses that sell to the M. D.'s, as the dose for a pointer dog and a man are the same and medicine is medicine, whether for a man or a dog, or any other animal, and as I have said at the beginning of this article, the practicing veterinarian has got to use economy nowadays if he expects to pay overhead expenses, compete with the quack, the patent medicine vendor, the farm agent, and "home doctoring," etc., but he certainly can't do this if he buys all these commercial houses that sell to veterinarians only have listed in their catalogues.

Mobile, Ala. G. W. Browning, V. S.

Parturient Paresis Remote from Parturition

ON PAGE 409, September issue (Volume XXIII, No. 9) of VETERINARY MEDICINE is an interesting article by J. S. Leslie, reporting a case of parturient paresis three months after calving.

The doctor asks if others have had a like experience. I wish to refer the doctor to an article I wrote on this subject in 1919, page 185, April issue VETERINARY MEDICINE, (Volume XIV, No. 4). I here quote in part:

"While by far the greatest majority of cases are so intimately associated with parturition, that is, occurring within a few hours to forty-eight hours after parturition, still occasionally it occurs before calving and not infrequently a considerable time thereafter. During the spring of 1906 I had seven of what might be termed delayed cases, that varied from one to three months after calving. These all occurred in cows that were on pasture during the wet spring season, when the pastures were unusually luxuriant and the milk flow had thereby been rather suddenly and considerably augmented. These animals presented all the symptoms that characterize the ailment and no other symptoms so far as our present knowledge of this disease will permit us to judge, and all responded to the usual treatment. This with subsequent experiences has proved rather conclusively to me that we may and do have cases of puerperal paralysis more remote from the date of calving than our present literature would indicate.

According to some, such cases are not true puerperal paralysis, but probably digestive paralysis. We will give our views on this question under "Differential Diagnosis." Others state that this condition does not occur at all before the membranes have been expelled. Obviously this cannot be true when it occurs before parturition, and my experiences have been otherwise, in some cases occurring after parturition.

I recall being consulted by telephone one evening relative to a sick cow, the history as given over the telephone indicating a case of parturient paralysis. Within a few minutes, before leaving my office, I received another urgent call to give attention to a horse suffering acute pain. On arriving at the place where the cow was I found a high-grade Jersey that had calved that morning, manifesting all the symptoms of parturient paralysis, the afterbirth retained. Without giving any attention to the membranes I hurriedly inflated the udder and explained to the owner the urgency of the call a few miles away and promised to return as promptly as I could to give further attention to the cow. I returned in about two hours and found the cow sitting up, quite conscious, with the uterus everted and the afterbirth adhering to many of the cotolydons. The membranes were removed, the uterus cleansed and returned to its normal position and the cow promptly recovered without further mishap. According to statistics collected by Jensen, in 1,107 cases, 4.24 per cent occurred before birth, and the remainder after birth. Of the latter

2.53 per cent began after one to three hours.

3.9 per cent after three to six hours.

13.09 per cent after six to twelve hours.

14.27 per cent after eighteen to twenty-four hours.

10.29 per cent after twenty-four to thirty hours.

8.40 per cent after thirty to thirty-six hours.

3.70 per cent after thirty-six to forty-two hours.

6.77 per cent after forty-two to forty-eight hours.

5.42 per cent after more than forty-eight hours.

Hess observed no cases occurring more than ninety-six hours after parturition, although there are records in the literature where the disease was said to have occurred much later, as long as ten days after parturition (Harms), but such cases may have been confounded with other diseases, as is suggested by the investigations of Guillebeau and Hess.

Harms, quoting Haycock, gives the following duration of time after parturition at which parturient paresis made its appearance:

5 times immediately after calving.
8 times 20 hours after calving.
5 times 23 hours after calving.
5 times 24 hours after calving.
3 times 30 hours after calving.
2 times 36 hours after calving.
1 times 42 hours after calving, etc., etc.

Since publishing this article, I have seen several other cases occurring as late as three or four months after parturition and some several weeks before parturition and I am quite sure that you have published similar experiences by other practitioners but I cannot recall just when.

Goshen, N. Y. J. DeVine.

Editor's Note: There have been a considerable number of reports published in VETERINARY MEDICINE which record the occurrence of parturient paresis in cows not only a short period before calving but considerable periods afterward, and in cases of difficult parturition, in abortion and in eversion of the uterus; all of which is contrary to the text-book teaching of a score of years ago and earlier. Among references to such cases may be cited case reports by the following:

B. W. Truax, a case occurring two months after calving, Vol. VII (1912) No. 9; O. H. Gebhardt, three months after, Vol. IX (1914), No. 1; J. F. Gillespie, three months after, Vol. IX, No. 2; O. W. Snyder, four days before and four weeks after, Vol. IX, No. 12; J. F. DeVine, cases three or four months after calving were common one spring, Vol. X (1915), No. 5; F. M. Long, one case eight weeks after and another five months after, Vol. X, No. 5; R. W. Regan, two months after, Vol. X, No. 10; M. R. Blackstock, eleven months after, Vol. XI (1916), No. 1; J. C. Callender, Vol. XI, No. 8; A. B. Sexsmith, many cases remote from parturition, Vol. XI, No. 9; and T. B. Hinkle 13 months after calving, Vol. XI, No. 10.

DIFFUSE HEMORRHAGE POSSIBLY SWEET CLOVER POISONING

Fourteen days ago a farmer found a horse near death in the pasture. It died immediately after having being found and I was called, and posted the animal, and well defined hemorrhagic lesions throughout the body were found. The remainder of the horses were given 5cc doses of hemorrhagic septicemia aggressin and they were removed from this pasture which is sweet clover and blue grass.

After about a week on blue grass pasture these animals were returned to the sweet clover and blue grass pasture again. They were back in the original pasture about a week when a black gelding nine-year-old and weighing about fifteen-hundred pounds was noticed sick and was taken to a shed nearby.

When first seen, this horse was walking with a stiff-legged gait, sweating profusely, and there was amaurosis and amasesis. The ears and tail were carried as in tetanus and there was accelerated breathing and a rapid pulse. The temperature was 101° F. This animal had a highly congested mucus membrane of the eyes, nose and mouth. The membrane of the eyes was of a yellowish color and nasal mucosa was hemorrhagic.

This horse did not respond to medicinal treatment and lived only about three hours after first being noticed sick.

A post-mortem examination was held after dark by the aid of light from an automobile. There were bloody infiltrations throughout the body. The heart and lungs were very hemorrhagic and there was a ruptured blood vessel in the medulla. All the blood vessels of the brain and spinal cord were very highly congested. —S. G. M., Iowa.

Is Canine Distemper a Danger To Children

By ARTHUR H. BRYAN, Baltimore, Maryland

MANY veterinarians will recall that during the world war, at the medical, dental and veterinary officers' training camp, at Camp Greenleaf, Ga., that during the terrible outbreaks of "Spanish flu" in 1917, canine distemper came into the spotlight as a possible etiological factor. Medical and veterinary officers working with bacteriologists discussed the possible relationship existing between Spanish flu and canine distemper, because both diseases had similar secondary invaders, with the specific character of the primary, exciting cause unknown. The symptoms of both disases, with the variety of forms, such as the catarrhal, pulmonary, nervous, and digestive, together with the pathological changes, were very similar.

Experimental inoculations were carried out but the results were inconclusive as were all serological and bacteriological researches carried out with both diseases. The decline of the Spanish flu epidemics left the disease amongst the "unknowns."

The common colds of the human, with its varying ramifications of coryza, influenza, bronchial catarrah, pneumonia, etc., all have etiological and pathological pictures very much like the varying forms of canine distemper, with the added uncertainty of the actual causative agent. In February 1927 and 1928 the writer was veterinarian to the Maryland Kennel club dog show, and following both shows I went down with a severe case of "La Grippe." In an effort to throw light on the inter-relationship existent between the two diseases, human and animal, I carried out some experimental inoculations of the heavy, yellow, muco-purulent discharges from my nose, and bronchial exudates expectorated by me, and injected the material directly into four mongrel puppies, all out of the same litter, aged about seven months, and all in perfect health. Within seven to ten days all four puppies went down with the typical picture of canine distemper, which included serous and later purulent conjunctivitis; rhintits and later muco-purulent discharge, two of them developed the exanthematous pustules on the abdomen so typical of distemper. Two died within a week of broncho-pneumonia, one case seemed likely to recover but developed the nervous form with its attendant twitchings typical of chorea, the other one went through all the stages of the catarrhal form of distemper and slowly recovered. The post mortem examinations of the three that died were characteristic of the usual lesions found in the respective forms of the disease.

These results are of course by no means conclusive, they are merely preliminary experiments, for a dozen and one questions may come up not easily answered, but these results certainly justify further research and inquiry in a scientific manner not possible to the practitioner.

Some clinical case observations were made by me in the course of routine practice which bear directly on the problem.

Case No. 1. A close neighbor of mine had a large American bull dog, and it developed the catarrhal form of distemper, with its attendant sneezing, nasal and bronchial discharges, conjunctival pus, and elevated temperature left no doubt as to the diagnosis. The owner let the case run several days before securing professional services and during this time the four children had been playing with and fondling the dog as usual, and even more than usual because their pet was sick. All four children went down with heavy colds and influenza. The children were sick while the dog was suffering from the bronchial form of distemper, the dog had been sniffling and sneezing all over the house, and without any doubt sneezing

directly into the face of some of the children. On one visit I made to the dog, the physician attending the children was present also; a comparative study of both diseases in the children and in the dog revealed a close similarity. The rooms in which the dog had been were disinfected, the children's clothes boiled, and the dog was isolated. All the patients recovered, none of them developing the more serious sequellae.

Case No. 2. Some very close friends and neighbors of mine, making close observation possible, had a police puppy dog vaccinated against distemper. Whenever I used the vaccine from one particular biological house, I seemed to have had results follow, and in this particular instance the dog went down with typical canine distemper shortly after the last dose of combined serum and bacterin had been administered. The course of the disease in the dog was rather protracted, lasting about six weeks, the dog dying in convulsions. The dog was kept, while sick, in the cellar, but the history revealed that the children had been playing with the puppy while sick. The three boys, aged from three to six, all went down with broncho-pneumonia, with varying severity. All the children recovered.

Case No. 3. A well-known Baltimore lawyer recently bought a Boston bull terrier. The dog became sick, and the case was diagnosed as the catarrhal form of canine distemper. Again the history of the case revealed the fact that the three children had made a regular playmate out of the puppy, and apparently paid for their affection by developing, during the time the dog was sick, tonsilitis, influenza, and severe colds with the attendant high fevers. The evidence the lawyer agreed pointed to the infection originating with the distempered dog, and sent the dog away.

Case No. 4. A police dog puppy, eleven months old, had a catarrhal, bronchial, and later digestive type of distemper. The course of the disease was short, a hemhorrhagic gastro-enteritis with blood stained fecal discharges, and hemoptosis from the lungs soon terminated the case. The early symptoms of the disease were those of the ordinary catarrhal distemper. The young school girl of about eleven years of age, who nursed her pet during his sickness, went down with a catarrhal rhinitis and terminated in a mild case of influenza.

The above cases all occurred in my immediate neighborhood; I was therefore able to keep closely informed as to what occurred.

Conclusion. These cases just cited might easily be mere coincidences, but one conclusion can be safely drawn from the above observations: The human physician or veterinary surgeon upon diagnosing a case of canine distemper where children are liable to infection, is justified in observing prophylactic measures to the extent of insisting that the children be kept away from the dog entirely, and from any portion of the premises that the dog might have infected, until thorough disinfection. Now that extensive research is being carried out particularly at the Rockefeller Foundation and at the Medical School, Johns Hopkins University, with regard to colds and allied infections, seeking to supplement the above with more definite data, is worth while.

The small animal practitioner can do more than any other group of professional men to throw light on this problem by taking note of all cases of distemper occurring where children play with the sick animal and following up the inquiries as to whether colds, grippe, bronchitis, or pneumonia follow in the children, and once again place the veterinarian in the role as essential to the health of a community. Should further statistics and research fail to supplement the foregoing deductions, then the filterable virus, together with the secondary invaders, causing distemper could be definitely relieved of all responsibility in human disease. While it is generally conceded that filterable viruses are not communicable between the various species, the secondary invaders, streptococci, staphylococci, *B. bronchi-septicus,* and *B. Pfeifer,* are found present in canine distemper and the human diseases allied to colds and influenza.

Sterility an Important Swine Breeding Problem

By A. T. KINSLEY, Kansas City, Missouri

IN order that the swine industry may continue in its present economic relation to other branches of animal husbandry, it will be necessary to increase the efficiency of production and control of disease. The many problems incident to the breeding, feeding and management of swine require the careful consideration and co-operation of the veterinarian and the producer.

One of the most important problems that confronts the swine husbandman is low production due to abortion and sterility in sows and the excessive loss of small pigs. Abortion, shy breeders and sterility are of frequent occurrence in swine. The loss of pigs at the time or soon after farrowing is frequently associated with some disease of the genital organs of the sow, although the boar is no doubt an important factor in some cases. Abortion has been prevalent but the pig losses are not all due to abortion.

Prevalence

Sterile and slow breeding boars are more common than is usually suspected, and to attempt to estimate the extent of sterility in sows would only be a guess at random. There has been an effort made to obtain definite information relative to the prevalence of sterility but the data collected thus far is not sufficient upon which to base a definite statemen. The condition is apparently equally prevalent in all breeds. It is probable that more sows become sterile after the production of the first litter, which may have been normally farrowed or may have been aborted, than in either gilts or older breeders. Some farms have been carefully surveyed on which 50 per cent of the sows were sterile. In other instances only a small percentage of the sows are non-breeders. It is the exception to find a herd of 15 or more sows that are all breeders. Sterility in sows is more or less prevalent in all sections of the country, but it would appear from the available information that it is most prevalent in those areas where there is intensive swine production.

Many practitioners contend that sterility is more common in young boars than in the mature animal.

Causes of Sterility

An occasional case of sterility occurs in gilts or boars as a result of improper development or malformation of the genital organs. Inbreeding long continued may be a factor in sterility. Insufficient exercise and overfeeding is a common cause of sterility, particularly in gilts.

It is probable that the most important factor in the occurrence of sterility in sows is the influence of previous disease. Infections of the genital organs that produce abortion frequently bring about changes that result in sterility.

Abortion and the production of pigs with a low vitality is a common sequel of swine "flu" and may also occur as a result of hog cholera, and in such instances there may be a secondary infection of the uterus resulting in sterility. Diseased ovaries is also an important factor in sterility.

Non-breeding or sterility in boars is usually the result of injury of the genital organs. Such injuries may be due to mechanical interferences and infection, but is frequently the result of excessive use or abuse. Small litters and those litters in which the pigs have little vitality can frequently be traced to pasture breeding or to a boar that has had excessive use.

Symptoms

The detailed symptoms of sterility in sows is common knowledge and suffice at this time to state that sterile sows may

or may not evidence estral periods. In some the estral period is regular, in others irregular, and an occasional sow is observed that is in heat continuously. A persistent purulent discharge is indicative of vaginitis or metritis. There may be a pyometra or purulent collection in the tubes.

Sterile boars may show disease or defects of the testicles. Boars with diseased genitals are usually unthrifty and show more or less disturbance of locomotion.

Prophylactic Measures

The production of swine can be increased by observing the primary principles of feeding, sex hygiene and sanitation. Breeding swine should not be given a fattening ration but should be provided with a properly balanced maintenance ration. In this connection the statements of Professor Meneough in his article on "Herd Management of Swine" in the October issue should be studied with the greatest care. Increasing the breeding efficiency of swine is a real problem. It begins with the feeding of the young gilt and the young boar. Professor Meneough's discussion of this subject is sound.

It is very probable that some of the pig losses have occurred because corn and water constituted the ration of the breeding animals. Ground oats or barley and semi-solid buttermilk is a splendid food for breeding sows or boars in service. The mineral ingredients in foodstuffs vary, but from the evidence now available it appears that some foodstuff is deficient in the essential minerals and it is advisable to use mineral substitutes.

Inbreeding and line-breeding are of value in fixing some peculiarity of the breed, but should not be continued until it results in diminished vigor and vitality. Breeding swine should not be kept in small pens but should be so maintained that they are forced to exercise. Excessive use of the boar may cause orchitis, which will diminish the vitality of the sperm cell and may destroy them. Boars should not be permitted to run with the sows during the breeding season, as this favors excessive service. All breeding should be done in a small pen and after one service the sow should be removed. A young boar should not be permitted to do more than one service every other day and a mature boar one service each day. The excess service in pasture breeding is probably responsible for a large percentage of the small litters.

Recent aborters or diseased sows should not be bred until all evidence of disease has disappeared. All aborters or other diseased sows should be segregated and properly cared for to prevent the spread of the infection.

Value of Treatment

The important question from the breeders' viewpoint is, shall the aborters and apparently sterile sows or boars be retained? An intelligent answer to the question involves a consideration of several factors. If a sow is a good individual then it is worth while to attempt to overcome the infection of the genital organs and prevent sterility. In attempting such treatment the sow should be isolated and the proper ration as previously indicated should be provided. A bacteriologic examination of the genital discharges has revealed a variety of microbes. Many practitioners have reported favorably on the use of one or more injections of a combined bacterin made from the microbian invaders and it seems justifiable to recommend its use in conjunction with carefully applied douches of non-irritating solutions. The percentage of sterility, according to the reports from practitioners, in sows that have aborted and in which the above treatment has been carefuly executed is very small and the expense of treatment is not prohibitive.

Treatment should not be recommended for aborting or sterile sows that are not good individuals. Such sows should be placed in the fattening pen and sent to market. The treatment of sterility in sows involves many different problems. In some cases this condition has been

corrected, according to reports, by providing a properly balanced ration and particularly the addition of mineral substitutes. This method of treatment will produce results only in those cases in which there is little or no alteration of the genital organs. The cases of sterility resulting from infection of the uterus may be overcome by the line of treatment suggested for genital infections following abortion. Sterility resulting from cystic ovaries or similar conditions may be overcome, in some cases in large cows, by manipulating the ovaries. The ovarian cysts are ruptured in the same manner as those of the cow. Cystic ovaries may also be treated by making an opening through the abdominal wall similar to the incision made for the flank method in Caesarean section. This method of treatment is radical and it is doubtful if it should be advised, except in extreme cases.

Sterility in swine is an important problem and one in which the veterinarian can render the swine industry a valuable service. From the available information it would appear to be more economical to treat aborters and sterile sows that are good individuals and have previously been good breeders than to depend upon gilts each year for pig production.

Polyarthritis of Swine

By A. T. KINSLEY, Kansas City, Missouri

POLYARTHRITIS is a disturbance of various articulations, characterized by a subacute or chronic inflammation of the structure of the joints. In swine this disease is essentially chronic, has an insidious onset and is usually not identified until the affected animals are slaughtered. The extent of this disease has been difficult to ascertain because few feeders or breeders recognize it, and only a few cases have been reported by practitioners.

The most reliable information available indicating the extent and distribution of polyarthritis are the reports of the postmortem findings on hogs slaughtered in packing houses. According to this information polyarthritis is more or less prevalent in swine in the north central states. The prevalence of the disease is apparently not confined to any particular type of breed of swine and occurs equally in males and females.

Polyarthritis of swine is primarily a disease of young animals, although if affected individuals are kept, the disease will persist in most cases throughout the life of the individual. It is of economic importance because affected individuals are unthrifty and do not develop and fatten normally.

Cause

Improper housing and, particularly, confining pigs on unbedded cement floors during cold weather is an important predisposing factor in the occurrence of polyarthritis. Severe weather during the winter is apparently conducive to this malady, for according to the findings at abattoirs there are proportionately more cases after long cold winters and cold springs than when the weather has been moderate or mild.

According to some investigations the *B. pyogenes* are frequently found in the lesions and are thought to be a primary causative factor of polyarthritis. It may be that this disease is a sequel of navel infection or at least has its origin at the time or soon after farrowing.

Symptoms

Polyarthritis is essentially a chronic condition. The onset is insidious. The early symptoms of the disease may be overlooked by the caretaker, particularly those cases in which the disease is not severe. The first manifestations of this disease are locomotory disturbances. In

the beginning the affected joints are usually swollen and sensitive. As the disease progresses the inflammatory process becomes subacute or chronic and the joints may or may not be enlarged. There will be variable interference with joint action, depending upon the extent and nature of the lesions. Affected swine are unthrifty; in fact, unthriftiness is sometimes the only manifestation of this disease.

Lesions

Affected animals may be underdeveloped, unthrifty and more or less emaciated, depending upon the stage and extent of the disease. The lesions characterizing polyarthritis vary according to the extent, intensity and chronicity of the process. In the early stages the synovial membrane is congested, and as the disease progresses varying quantities of serous fluid accumulate in the joint cavity. In mild cases there is limited inflammation and infiltration of the synovial membranes and perisynovial structures. The serous fluid that accumulates in the joint cavity is usually turbid and may contain flocculi.

In the later stages, particularly of the chronic cases, there is a thickening of the synovial membrane due to fibrous, proliferation. Fibrous formation may involve the perisynovial structures and be sufficiently dense to render the joint immobile or nearly so. The articular cartilages may be eroded and there may be organization and fibrous proliferation within the joint cavity, thus limiting or inhibiting movement. Lesions typifying complications of various other diseases may be noted.

Treatment

Treatment for swine affected with polyarthritis may be of value in the very early stages. Unfortunately the disease is frequently not recognized until the latter stages, at which time treatment will be of little or no value. During the acute inflammatory stages the disease may be temporarily relieved and in some cases overcome by the application of hot packs to the affected joints. This treatment is not practical except in the case of high-priced breeding animals.

Polyarthritis is reduced to a minimum by breeding only healthy sows; by providing sanitary surroundings for sows during farrowing; and by properly housing pigs and shotes and providing proper bedding to prevent contact with cold, damp floors.

MOLDY FODDER INJURIOUS TO CATTLE

Dr. A. L. Wood of Hampton, Iowa, writes as follows:

"I am forwarding you today a sample of corn fodder (shredded) that I would like to have examined, as I noticed it is well covered with white mold and I remember you told us that some molds are poisonous. I have attempted to read this up in your "Manual of Poisonous Plants," but can't say that I have accomplished much.

"This corn was in pretty good condition when shredded, so owner informs me, but he sure has a mess now. The middle of the pile is wet and steaming and resembles a manure pile more than anything else. The owner is an Ames man that took C. E. course. Will you please advise me as to the chance of trouble if owner continues to feed this fodder. I treated his cattle for hemorragic septicemia with the aggressin some time ago but do not think this will protect them against the white mold poisoning should this be the fungus that is poisonous."

The specimens sent were very moldy and through the kindness of Dr. J. C. Gilman, these molds were submitted to a study by making cultures. He found three types of mold present, the Acremoniella, Cephalosporium, and Cephalothecum, all more or less common molds. There is no question in my mind but that when mold is as abundant as it is in the sample sent, that some trouble will be caused from irritation of the mucous membrane of the stomach and intestine. They are injurious and under no conditions should fodder as moldy as this ever be fed.

Ames, Iowa. L. H. Pammel.

Hog Cholera with Special Reference to Differential Diagnosis

By A. EICHHORN, Pearl River, New York

IN RECENT years veterinarians have been confronted with difficulties in the differentiation of the various infections in hogs. This problem has become so general that those engaged in the control of swine infections are frequently at a loss in establishing a definite diagnosis. Thereby they are confronted with situations which make them uncertain as to the procedure of control.

After the early investigations on hog cholera, it was generally assumed that practically all infections in hogs were due to the filtrable virus and the control of the infection by vaccination has been highly successful. In fact, the successes attained from the protective vaccination of hogs against cholera were so conspicuous that the incidental failures were not given serious consideration.

Investigations have disclosed the fact that in the majority of cases the losses resulting among vaccinated animals are due only exceptionally to the filtrable virus, but in the majority of cases they are the result of some complication with the so-called mixed infections. Furthermore, they have also established the fact that there are infections among hogs which are independent of the cholera virus.

In order that losses from infections in hogs may be minimized, it is primarily essential that we gain a thorough knowledge of the manifestations of the different infectious diseases in hogs which would enable us to make an accurate differential diagnosis for the purpose of intelligent control work. Hog cholera caused by the filtrable virus must be recognized as being by far the most frequent infection and the one causing the greatest destruction in hogs. The appearance of other infections must be now considered a sequel of hog cholera in all cases in which an independent occurrence of these infections is not established. Independent outbreaks with pathogenic organisms which were heretofore justly considered only as secondary invaders in association with virus cholera must be, however, given due consideration.

Veterinarians who have studied hog infections for the past fifteen years recognize the fact that conditions have arisen which were not present in the early days of hog cholera control. The so-called secondary invaders have apparently developed an increased virulence. By passage through animals the other infections of hogs may have gained an increased virulence and become by far more pathogenic than they were in the past. Another reason the so-called mixed infections are occurring now more frequently than in the past, may be due to the spread of the infection by so-called carriers. Carriers are now recognized as the greatest menace to disease control by sanitary police authorities.

Carriers in animals are recognized as big factors in spreading contagions. Thus normal animals may harbor the infection and at favorable opportunities communicate it to susceptible animals. Influenza, dourine and foot and mouth disease have been proved to be sometimes disseminated by such carriers.

In mixed infections of swine we have three groups or organisms especially responsible for infections and the characteristics of all these organisms suggest the probability that they may be present in healthy individuals—the so-called carriers.

Bacteriological studies of the bacterial flora of the respiratory and digestive tracts of hogs have shown that the hemorrhagic septicemia organisms, the *B. suipestifer* and the *B. paratyphosus* are present in apparently healthy hogs.

Finally one must not lose sight of the pos-

sibility that the extensive use of vaccination for the prevention of hog cholera has probably resulted in increased resistance in our present hogs and that as a result of this, the disease caused by the filtrable virus becomes somewhat obscured, and the secondary infections, which have always been recognized in connection with hog cholera, appear to be predominating. As a result our attention becomes distracted from the true etiological factor and in the absence of the typical cholera lesions the veterinarian is apt to make a diagnosis of either hemorrhagic septicemia or necrotic enteritis, whereas actually the true original factor is the filtrable virus.

I have abstained from mentioning the so-called necrobacillosis since the introduction of this name has never been justified as there never was sufficient scientific data presented by which the *B. necrophorus* could have been incriminated as being the primary responsible factor for the intestinal necrotic infections of hogs. On the other hand, artificial infections with *B. suipestifer* and *B. paratyphosus* have successfully produced lesions commonly met with in intestinal infections of hogs. Thus the presence of the *B. necrophorus* at the best can be accorded only of secondary importance in such lesions.

It is not the intention to discuss the infections caused by germs pathogenic to hogs, but it might be advisable to emphasize the fact that the lesions produced by them are occurring now with greater frequency and that their significance should not be underestimated. The control of these infections, however, is essentially a control of hog cholera and an effective and successful control of hog cholera would result in a very pronounced diminishing of the other infections.

Hog cholera control has been exhaustively treated in bulletins and scientific articles appearing in our journals. The disease and its prevention is familiar to every one interested in the treatment of swine diseases; yet on account of the variations of the symptoms and lesions disclosed by the animal affected with the disease even the most experienced veterinarian is at times at a loss to make a definite diagnosis either from observation of symptoms of a single or several postmortem examinations. This is, no doubt, due to the fact that the disease manifests itself in many different forms. In some instances it is peracute; other times subacute, and may even develop into a chronic form. The symptoms and lesions vary accordingly and become more or less obscure, and due to this fact the uncertainty of a definite diagnosis by the veterinarian is so common.

Unfortunately, aside from the clinical and pathological manifestations and test inoculations we have no means at the present time by which the diagnosis of hog cholera can be made. There is no reliable microscopical, cultural or serological test known which would be of assistance in the diagnosis of cholera. The test inoculations, for which purpose only pigs can be utilized, offer only a slight assistance on account of its expense, laboriousness and length of time it required. Due to these facts, it is essential to make the most careful observations and examination of the animals suspected to be affected with hog cholera and only by studying the cases in detail will it be possible to reduce failures of diagnosis to a minimum.

In considering the symptomatology and morbid anatomy of hog cholera, it is possible in most cases to make a diagnosis of hog cholera with certainty and without much delay, especially in cases where a number of animals are affected in a herd. Only in cases where the symptoms and lesions are not pronounced is a mistake in diagnosis excusable. Clinical observations alone can lead only to a probable diagnosis. The most important symptoms for a clinical diagnosis of hog cholera are general systemic disturbances, persistent or intermittent diarrhea, conjunctival catarrh, papular exanthema with focal hyperemias or hemorrhages and necrosis of the skin. If the majority of these symptoms occur during the course of the disease in a single animal, the diagnosis of cholera may be made with all prob-

ability. Such cases, however, are usually only exceptional. Fortunately, in most instances there are several animals affected in a herd and by grouping the symptoms as they occur in the different animals, a definite diagnosis may be made.

If the clinical manifestations are not sufficient to insure a diagnosis, a definite decision may be made by post mortem examination on an animal already dead with the disease or killed on account of the disease. In such cases, a diagnosis of hog cholera may be made from the presence of the lesions such as the characteristic hemorrhagic gastro-enteritis which usually occurs in association with multiple hemorrhages in the kidneys, or the serous membrane of the lungs and in other parts. In acute cases the inflammatory swelling of the lymph glands is of great diagnostic value. In animals which are killed during the period of incubation or during the first days of the disease the involvement of the lymph glands is frequently the only demonstrable change found on autopsy. Associated with the lesions in the lymph glands we most frequently find the petechia, especially so under the pulmonary pleura. Of the lung lesions those which show a tendency towards extensive hemorrhages into the pulmonary and interstitial tissues of the lungs are indicative of hog cholera. Multiple lobular, or serohemorrhagic pneumonias are specific for hog cholera. If the postmortem examination of a single animal is not sufficient to make a decision, a further observation of the disease and autopsies of other animals will in most cases lead to a definite diagnosis. In such cases, however, the treatment should not be deferred, as delay in waiting for more definite signs may prove costly.

The greatest difficulties in the differential diagnosis result from the colon-typhoid infections of hogs and from hemorrhagic septicemia. Other infections are of slight importance as the manifestations in such cases are very pronounced and not likely to be confused with hog cholera. Such are especially anthrax, scours in pigs, necrobacillosis in young pigs, tuberculosis and parasitic infestations.

The colon-typhoid infections of hogs, which are manifested in the form of necrotic enteritis, were prior to the discovery of the filtrable virus classed as cholera infections. Later it was found that in some outbreaks which closely resembled cholera the filtrable virus could not be demonstrated, but bacteria of the paratyphoid group were readily isolated, with which it was possible to produce in test pigs a disease identical with the cases occurring in the outbreaks. Thus it has been observed that the colon-typhoid infections occur independently of the virus cholera but not as serious outbreaks. The course is usually chronic and the mortality much lower than in hog cholera. Diarrhea is usually persistent but not intermittent as it is frequently observed in cholera. Still more marked are the differences found on autopsy. The principal changes are found in the large intestines and consist of a pronounced smaller cellular inflammation of the mucosa and submucosa resulting in a considerable thickening and a peculiar dense, tough consistence of the intestinal wall. In the more chronic cases which are the most frequent, the lymph follicles are first to become swollen. The swelling is so pronounced that there is necrosis of the follicle and an extension of the process to the adjoining tissue. The necrotic lesions in the colon-typhoid infections are of a caseous-diphtheritic inflammation which results in various sized round comparatively even or irregularly contoured ulcers.

In the bacteriological examination of the colon-typhoid infections of hogs, it is not always possible to cultivate the same organisms. The organisms, however, which are responsible for these lesions have been found in most instances to belong to the paratyphoid group and some of the infections have also been traced to the *B. suipestifer*. For a differential diagnosis, however, the demonstration of any of these bacilli should not be considered as conclusive as

under certain conditions these germs may be present in hog cholera as secondary invaders. The principal stress should be laid upon the symptoms and the autopsy findings.

Hemorrhagic septicemia, which is caused by the *B. suicepticus* and which constitutes principally an affection of the respiratory organs, is primarily brought about by conditions which have a tendency to predispose the respiratory organs to the infection. Under such conditions the hemorrhagic septicemia organisms which are so widely distributed will readily attack these predisposed organs. The affection is distinguished from hog cholera by the fact that it usually runs a more chronic and milder course and furthermore by the absence of symptoms of the digestive apparatus, whereas the involvment of the respiratory apparatus are predominating. Besides, in hemorrhagic septicemia the skin shows no changes with the exception of a non-specific diffused crustaceous-eczema and a cyanotic discoloration. More characteristic from a standpoint of differential diagnosis are the postmortem findings. In most instances in hemorrhagic septicemia and the affections simulating hemorrhagic septicemia we find lesions in the respiratory apparatus, whereas the stomach and the intestines show no changes. Exceptionally a gastro-intestinal catarrh with circumscribed hemorrhages may occur in isolated cases. In the presence of such catarrh the surface of the inflammatory parts may even show fibrinous exudate and the superficial layers of the mucous membrane may show a necrosis as if sprinkled with bran. Necrosis of the deeper portions and above all the diphtheroid inflammation are never present. The fact that in some cases hog cholera may show manifestations of hemorrhagic septicemia cannot be denied and in such instances it is necessary to make further observations of the infected animals in order to make the differentiation possible.

The bacteriological examination does not play an important part in the differentiation between hog cholera and hemorrhagic septicemia since in hog cholera we may find the same bacteria in the inflamed lungs as in hemorrhagic septicemia. In the more protracted cases of hemorrhagic septicemia, the character of the lung infection represents a catarrhal pneumonia, whereas in the more acute cases, which are less frequent, we meet with the fibrinous pneumonia permeated with necrotic foci. In some outbreaks such acute fibrinous pneumonias may predominate and cases may even occur in which the manifestations are those of an acute septicemia without pronounced lung involvements. The bacteriological findings may show the presence of the *B. suisepticus* not only in the affected organs but also in the blood and furthermore in some of these cases it is impossible to establish the presence of the filtrable virus which appear to show conclusively that independent outbreaks of hemorrhagic septicemia do occur. Nevertheless, we must assume at the present time that they are infrequent and represent only a small percentage of the losses in hogs.

We should not lose sight of the fact that severe infectious diseases aside from cholera occur only infrequently in hogs and in the majority of cases the presence of a combination of the symptoms and lesions points to a mixed infection with cholera. Hog cholera in itself is a disease of mixed infections, that is we will observe only in exceptional cases symptoms and lesions truly typical of virus cholera. In the great majority of cases, the autopsy reveals various kinds of mixed infections in association with the virus septicemia. The veterinarian should study every outbreak of diseases among swine and in the presence of any symptoms and lesions characteristic of cholera he should not hesitate to proceed with the control of the virus cholera and only in those instances in which there is a total absence of symptoms and lesions of this disease or in instances where so-called breaks occur among animals which have been properly vaccinated with a potent anti-hog cholera serum and virus should he proceed with the differential diagnosis and then based on his findings he should decide upon the control of the infection.

Some Plants Poisonous to Swine

By L. H. PAMMEL, Ames, Iowa

IN THIS brief account of plants that poison swine, it will be impossible to give details. We will use the subject of poison in its broadest sense.

Swine are very generally believed to be quite tolerant of certain poisons harmful to other animals. Thus, they are regarded as being immune to snake venom, or at least rattle snake venom. It is widely believed that they are not affected by botulinus toxin or other toxins occurring in moldy or putrified vegetable or animals products. These impressions and the fact that hogs do not live on green plants to the extent that cattle and horses do, have perhaps led swine raisers in many instances to suspect other agencies as being the cause of death in cases of true plant poisoning in hogs.

That a very considerable number of plants common in the fields and pastures and in the hog lots of this country are poisonous to swine when eaten, has been amply substantiated by field cases and controlled experiments. It is probable that healthy hogs that are properly fed, seldom eat injurious plants; but, on the other hand, it is also certain that in the early spring when green forage is scarce, in cases of malnutrition, and when kept in dry lots and fed an unbalanced ration, hogs do when opportunity offers, occasionally ingest plants that result in poisoning and death; and that the loss each year from plant poisoning in swine in the aggregate amounts to a very large sum.

An examination of the works of H. Casparius,[1] K. Glasser[2] and W. Pfeiler[3] shows that a great many more cases of plant poisoning in hogs are recorded in Europe than in this country. Since the number of poisonous plants are approximately equal in the two countries and the conditions under which hogs are raised exposes them at least as much in this country as in Europe, this seems a further evidence that many cases of plant poisoning in this country are not reported, and probably are not suspected of being due to their true cause.

Cockle-Bur Poisoning

Cockle-bur poisoning is reported more frequently than any other of all other plant poisoning of swine. Kinsley[4] lists comparatively few plants that are poisonous to hogs, but gives the cockle-bur first place. Dykstra[5] states: "Every spring the Division of Veterinary Medicine receives numerous reports of death among pigs. . . . The stomachs of the dead animals are loaded with young cockle-bur sprouts."

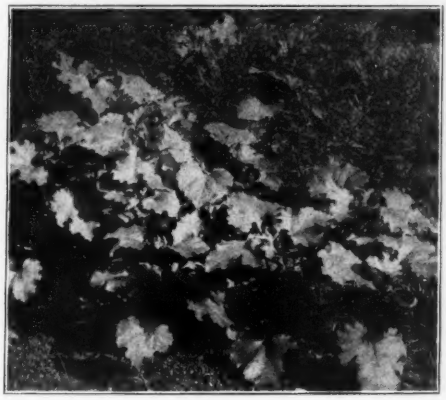
That cockle-burs in the cotyledon stage, that is just before the first pair of true leaves appear, is a frequent cause of poisoning in hogs in the early spring is amply substantiated by the experiments of Hansen, Marsh, Cary, Kinsley and others, and by field observations by a large number of veterinarians. In eating these young cockle-bur sprouts, pigs frequently eat the burs also, and these cause mechanical injury to the stomach and intestines, sometimes being the cause of death. However, death from eating young cockle-burs is ordinarily due to a poison in the green plants.

Kinsley in his book on "Swine Diseases," reports on some expriments conducted with cockle-burs. He states that he made an investigation of the effect of young cockle-burs to swine. He gave some young cockle-bur plants to pigs weighing from 40 to 60 pounds and in all cases there was indication of gastro-enteritis and that it involved the entire mucosa of the stomach and small intestines and not infrequently extended to the mucuous membrane of the large intestine. The symptoms were increased thirst, colic pains, diarrhea, weakness, paralysis, and death succeeded rather rapidly in fatal cases.

Dr. R. R. Dykstra[5] of the Kansas State Agricultural College, in a recent circular, states: "Every spring the Division of Veterinary Medicine receives numerous reports of deaths amongst pigs. In some of the reports, the owner is entirely at a loss as to the cause of death, because he will state that the animals have received no change of feed or care and therefore it is a mystery to him why several of them are found dead in the hog lot. A careful inquiry into the handling of the hogs usually discloses that, a day before the deaths occurred, the animals had been given access to a lot through which passes a small creek or other stream, that the water in the stream is very low and that for several days past the weather has been more than ordinarily spring-like. This warm weather, combined with the necessary moisture requirements in the bed of the stream, has resulted in a quick sprouting of the cockle-bur. When areas of this nature are examined, they will be found to be literally covered with young tender cockle-burs that have just made their appearance through the ground. At this stage of its growth, the young cockle-bur is appetizing, the pigs will eat almost anything of a green nature, so that the stomachs of the dead animals are loaded with this material."

A fine paper has been published on cockle-bur poisoning by C. Dwight Marsh, Glenwood C. Roe and A. B. Clawson.[6] From these experiments there can be no doubt as to the poisonous character of cockle-bur and other species of Xanthium. They describe the symptoms as follows: "First symptoms noted in poisoned pigs, ordinarily, was depression. This was accompanied in most cases by nausea which frequently resulted in continued vomiting. As the sickness progressed the animals became so weak as to be unable to stand. In those that were very sick the respiration became labored, the animals frequently gasping for breath. The pulse became rapid and weak. In some cases there were spasmodic movements of the body. Occasionally the animals moved their legs in what might be described as running movements. Sometimes, however, death resulted without these spasmodic movements."

In their investigation they found that the walls of the stomach of the pigs were more or less congested. This congestion extended, in some cases, to a greater or less degree, to the duodenum, jejunum, ileum, cecum, and colon. Where this congestion occurred there was a tendency to thickening of the walls with a serous infiltration. They found the toxic doses to be as follows: "As is shown elsewhere, the plant was found to be poisonous only in the younger stages; the largest part of the experimental work was done, therefore, on that stage of the plant which has been designated as the young cotyledon stage. This means the stage of the plant up to the time the first pair of leaves is partially formed. In gathering the plant for experimental purposes, especially during the later feedings, great care was taken to get it as young as possible. Plants in which the first pair of leaves were much developed were not ordinarily used. In the following discussion of dosage only plants of this age are considered.



Xanthium Canadense (cocklebur)

"In general, it may be stated that the lethal dose for pigs is about 1.5% of the weight of the animal, and that the toxic

dose may be as small as half of that quantity."

Dykstra states: "Research work has demonstrated that young cockle-burs are poisonous at the time the first pair of leaves are partially developed, or just after germination.

"Pigs that have consumed the young cockle-burs in this stage may vomit, they are depressed, and before death spasms may occur, though some animals die quietly.

"There is no specific remedy against cockle-bur poisoning once the poison is absorbed. However, if the hogs are observed eating these plants, they should at once be taken away from them and some fatty substance should be given to them. If hogs can be induced to drink whole milk, this apparently contains enough butter fat to very largely control the poisonous action of the plant. Other fatty substances, such as unsalted lard, and linseed oil, posses properties which stop the poisonous action of the plant."

Dr. F. R. Ahlers, Lamonte, Iowa, writes as follows: "It has been my observation that the plant of most interest in swine poisoning is the common cockle-bur. It seems to be most dangerous to very young pigs, only about six weeks old. The plant appears to be dangerous only when it is first appearing in the spring. Affected pigs lie prone or flat on their sides, as a rule, and keep all four legs in motion simulating a trotting or running motion. The muscles seem to be in a tense state. Excepting the movements of the legs, and the absence of spasm of the membrana nictitans, and the general muscle spasm, the cases are apt to remind one of tetanus in pigs."

Dr. J. A. Flinn, Jesup, Iowa, says, "The symptoms produced by feeding the young leaves of cockle-bur are convulsions and spasms. The usual course of the disease is about 12 to 24 hours. If large quantities are consumed the pig may die in a shorter period. Treatment is not entirely successful, but a heavy dose of oil is indicated not only for the laxative effect, but for the coating of the intestinal canal to prevent absorption."

Cockle-bur is widely distributed in the United States from the Gulf of Mexico to Canada and clear across the continent. The species, however, are not all the same. The common cockle-bur is a coarse, rough annual from one to three feet high; stems marked with brown spots; leaves rather large and heart-shaped with a long petiole; two kinds of flowers, staminate and pistillate, on the same plant in different heads; fruit covered with prickles and is two-beaked. The bur contains two so-called seeds.

Chemists have found that the plant contains the gluco-side Xanthostrumarin which resembles the substance datiscin; and, when heated, gives off the odor of succinic acid. It is rather strange that most of the poisoning occurs from eating the young plants, although the ripe fruit often produces irritation and causes mechanical obstruction.

Jimson Weed Poisoning

This is an extremely common weed in many parts of the state and in many places in northern United States. There are two species rather common, *Datura stramonium* and *Datura tatula*. These are rank-scented, tall narcotic herbs with funnel-formed white flowers; both are annuals. The stramonium has a purplish stem while the other has a greenish stem. Both of these species contain alkaloids known as atropin hyoscyamin, and hyoscin. There also is a so-called daturin which, however, is a mixture of hyoscyamin and atropin.

Jimson weeds are commonly found in vacant lots, especially in hog lots. Many farmers hold to the superstition that jimson weeds growing in a hog lot ward off hog cholera.

The symptoms of poisoning are vertigo, pain in the bowels, vomiting, nausea, extreme thirst, dry burning skin, nervous condition confusion dilated pupils, loss of sight, sometimes mania, convulsions, frothing of the mouth and death.

The experience of the writer is that hogs seldom eat the plant; but, as Dr. C. A. Cary of Alabama says, cases of swine poisonings from the jimson weed have been reported. Probably the seed, which does not have the

rank odor of the rest of the plant, is the part eaten and causes the poisoning.

Albert Hansen[7] also has discussed jimson weed poisoning in hogs. A number of cases occurred of which 3 died and G. E. Norman found the stomachs packed with the foliage and fruit of the jimson weed.

Black Henbane

This is another plant which is recorded as being more or less poisonous and which belongs to the same family as the jimson weed, which is the potato family. *Hyoscyamus niger,* Black Henbane, is a clammy pubescent biennial or annual which grows from one to three feet high with ovate sinuate toothed leaves. Flowers are on short stalks, dull yellow in color with purple veins. This weed has been commonly naturalized in some sections of this country as in Utah, and in some places is found from New England to Iowa. It contains hyoscyamin and hyoscin. This is one of the most poisonous of our plants. It causes spinal paralysis, retards heart action, loss of muscular power, stupor, dilation of pupils and death occurs from paralysis of the respiratory centers.

Night Shade

It is a mooted question whether the black nightshade, *Solanum nigrum* is poisonous. I am of the opinion that it is. The berries are black, the flowers are white. The leaves are ovate and stalked, with a rather strong odor. The plant is commonly naturalized in the United States. There are some undoubted cases of hog poisoning on record in this country.

The Western nightshade, known as the three flowered nightshade is similar to the above, but is much more toxic in its action. The flowers are whitish, the berries are black and occur three in a cluster. This nightshade is much more liable to cause poisoning to hogs than the common black nightshade.

Kinsley reports on nightshade poisoning as follows: "Poisoning is not frequent but it has been reported in parts of the country where these plants grow. Animals that die within a few hours will show no lesions at all except retention of the urine or a greatly distended bladder. The blood may be somewhat darker than normal. The first symptom noted is nausea, followed by vomiting, dryness of the mouth, more or less excitement and convulsions, increased pulse and respiration, dilated pupils and partial blindness. The duration of the period is short and it is followed by paralysis.

"Treatment consists in administering antidotes of atropin, emetics and purgatives; in stimulating the heart and restoring respiration by artificial means. In some cases sedatives may be given."

Cicuta maculata (cowbane)

Green Potato Poisoning

A number of European works mention poisoning from green sprouts. Cases of poisoning from eating potatoes with green skins are not infrequent in man. One of the more recent cases reported are those given where Albert Hansen calls attention

to fatal cases of poisoning in Illinois in which all the members of a family who ate the potatoes died. The symptoms recorded are colic, acute gastritis, nausea, enlarged pupils and diarrhea.

Most of the cases in hogs reported in this country were places in which garbage was used. There are many cases of this kind on record and doubtless many cases of death in garbage fed hogs due to potato-poisoning are attributed by the feeder to cholera, soap powder poisoning, broken glass, etc. Garbage frequently contains green (sunburnt) skins of potatoes or potato sprouts.

Ahlers also reports potato top poisoning in Iowa.

Casparius in his work, calls attention to poisoning from potato sprouts and that this is a common occurrence. It is also mentioned by Glover in Colorado.

Belladonna

The *Atropa belladonna* is sometimes cultivated in the United States and generally recognized as an extremely toxic plant to hogs by European veterinarians may be looked for as the cause of poisoning to hogs where the plant is cultivated. Dr. R. A. Craig of Purdue University reports enteritis from the matrimony vine, *Lycium halimifolium.* It has been reported to me as poisonous.

Cowbane, Spotted Cowbane or Musquash Root

Quite a large number of the plants of the carrot family are poisonous. Mention may be made of the poison hemlock *Onium maculatum* which poisoned Socrates. It is widely naturalized in North America from New England to Iowa, Utah and California. Water parsnips, *Sium cicutaefolium, Cowbane, Oxypolis regidior,* all with white flowers in umbels resembling wild carrot or Queen Ann's Lace for which they are mistaken.

I have recorded a number of other cases of poisoning from cownbane or spotted cowbane in VETERINARY MEDICINE. Poisoning from cowbane in hogs, perhaps, is of more frequent occurrence than is usually supposed, especially in some parts of the country where the ground is low. Our native cowbane is a tall, erect, branching herb, with hollow stem and large compound leaves and white flowers in umbels. It has a pungent odor. Roots are fleshy fascicled.

There are several other related species. All toxic. One of these, in eastern North America, has little bulblets and, another in the western United States, known as the water hemlock, has erect, horizontal roots with thickly fleshened horizontal root stocks which are chambered. It is most common in the Rocky Mountains and in Oregon. The cowbanes are the most deadly poisonous plants in North America. Many cases of human poisoning are recorded and some cases of hog poisoning. The poisonous substance is circutoxin. Violent convulsions occur with death occurring rapidly. I have found that the greatest danger is from the roots in the fall, winter and early spring.

Albert Hansen[8] has also reported some interesting cases. He has recorded 50 cases of poisoning from poison hemlock in cattle, hogs, sheep and horses.[9]

Mandrake or May Apple

It has long been known that the common May apple *Podophyllum peltatum* is more or less toxic. A perennial herb with running, yellowish rootstocks, a large peltate leaf and white flower, and a yellow fleshy fruit when ripe. The rootstock and the green leaves contain several poisons, one podophyllotoxine. The poison hastens biliary secretions, causes hemorrhoids, nausea, vomiting, purging colic, salivation and retching pains. Dr. J. B. Gingery, who reported on poisoning hogs in Muscatine County,[10] reports that five hogs died which had partaken of the leaves and stems of the plant. The animals had convulsions, staggering gait and rapid respirations. Postmortem examination of kidneys showed a petechia somewhat resembling the petechia of cholera.

Red Root

Red root, *Lachnanthes tinctoria* of the blood wort family and closely related to the lilies, is quite universally regarded as a poisonous plant in the south. It is a stout, tall herb, with numerous yellow

flowers, occurring especially in salty swamps from Massachusetts to Florida. It is quite generally believed that white hogs are particularly subject to the poison contained in this plant. White hogs are said to be killed off very easily while black hogs are said to be more or less immune. Certainly, it is conceded that this plant is poisonous to hogs in the South but it needs to be demonstrated whether white hogs are more susceptible to poisoning than black hogs.

Poke Root

Poke root, known botanically as *Phytolacca decandra,* is also known to have poisoned hogs. Hansen reports that some hogs in Indiana were poisoned by it. This plant is known from New York through Ohio, Indiana, Southern Iowa, Missouri and Kansas, and westward to Texas and east to Florida.[11] The berries are crimson and flowers greenish white and with large petioled leaves. Stems and leaves more or less succulent, coming from a long stout tuberous root, blackish in color.

Cockle and Daubentonia Poisonous

Quite a large number of plants of various families may produce hog poisons. A. L. Shealy and E. F. Thomas[12] call attention to the poisoning of poultry from the seed of *Daubentonia longfolia,* a commonly cultivated plant in Florida. It is possible although not proved that it causes poisoning in hogs. It has long been recognized that the seed of corn cockle, *Agrostemma githoga,* is toxic to hogs. Cases of poisoning of this kind came under my observation in Wisconsin many years years ago. Dr. H. Casparius of Germany lists this as one of the most common causes of poisoning in Germany, and where wheat screenings are fed in this country cases of poisoning are likely to occur. Since corn cockle is a common weed in grain fields it is likely to occur in wheat screenings. The toxic principles are sapotoxin and saponin. The symptoms are colic and vomiting, slobbering, bloody diarrhea, paralysis, hyperemia of the brain and a sharp, burning taste. Death is ultimately caused by the dissolution of the red corpuscles.

China Tree or China Berry

In an address on Florida poisonous plants made at the Florida State Veterinary Medicine Association in 1927, I referred to poisoning of hogs from the China berry, *Melia azedarath,* as reported to me by Fr. Fish at Gillis. In the same issue of VETERINARY MEDICINE in which my address was published, Dr. H. C. Nichols reports several cases of hog poisoning in Florida. The autopsies revealed an abundance of China berries in the stomach of the dead animals. When the remaining animals were removed to another pasture, no further deaths occurred.

Dr. P. W. Burns of College Station, Texas, carried on some experiments with China tree *Melia Azedarach* which I had previously reported as poisonous in VETERINARY MEDICINE. This tree is commonly cultivated in the south. It has compound leaves and purple flowers and a leathery fleshy drupe. Hogs frequently have access to the plant and no doubt frequent cases of poisoning occur. Doctor Burns has found this to be the case. He describes the symptoms of poisoning as follows: "I ground one-half pound of green fruit to a pulp and mixed with the feed. The pig, ninety pounds weight, ate two-thirds of this mixture one evening at 6 o'clock, and while I noted no symptoms at 12 m., at 6 a. m. it showed a marked intoxication with progressive weakness until death at 8:30 a. m. This pig could walk at a staggering gait at 6 a. m., but was down and could only crawl at 7:30 a. m. At 8:30 it was on its side kicking and unable to rise, and some of the muscles over the shoulders, sides and hams showed a marked twitching."

Lupine Poisoning

In the west there may be some cases of poisoning occasionally from eating the seed and pods of several species of lupine. The seed of the silvery lupine, Lupinus *argenteus,* according to O. A. Beath,[13] is known to be quite toxic, especially to sheep

and less so to hogs. He found some alkaloids. He found that when hogs were given slight doses they recovered promptly. L. W. Durrell and George H. Glover[14] note that it affects sheep chiefly. They note that the seed is likely to get into fodder at lower altitudes. Animals become frenzied, with violent spasms. Generally with a jaundiced liver.

Dog Fennel, Tansy and Fetid Marigold

Dog Fennel is well known to every one and needs no description. The flowers are in heads, with white ray flowers, divided leaves with a strong pungent odor. Cattle and hogs generally avoid the weed. Prof. P. P. Adams of Iowa City told me of several cases of poisoning reported to him. The owner lost 20 hogs which had been on dry feed. Turned out into a pasture. The plant acted as an irritant. The Fetid Marigold, *Dyssodia papposa,* and Tansy, *Tanacetum vulgare,* related plants are injurious in a similar way. The latter causes mental confusion, dilation of the pupils. It is a common weed in Northern United States.

Cherry and Plum Pits

The bulletin by C. E. Fleming, M. R. Miller and L. R. Vawter, [15] gives some interesting details about poisoning from the western choke cherry. Occasionally hogs may be poisoned by this cherry. And the European authorities like W. Pfeiler, Karl Glasser and Casparius mention poisoning of hogs from members of the genus prunus. Much has been written on the subject of poisoning from prussic acid. Several members of the genus prunus, sorghum, Johnson and Sudan grass cause prussic acid poisoning. The danger of this kind of poisoning to hogs comes largely by consuming the seed of the wild black cherry, choke cherry and peach pits. There is little danger to hogs from feeding on wilted leaves. With this poisoning there is a depressing action on the nervous system, the brain and spinal cord become paralyzed, convulsions occur, and the blood, at first dark, becomes lighter in color.

The Castor Oil Plant

The castor oil plant is commonly cultivated in some sections of the United States and is a frequent escape from cultivation from New Jersey to Florida and California. Occasionally hogs have access to material from which the oil has been removed. The ricin in the castor oil bean is an extremely toxic substance. Glasser makes mention of the castor oil bean, as well as the annual Mercury of Europe, which is an occasional escape in this country and is known to be toxic to hogs. It is a member of the same family. The several species of Croton, some of which are weeds, produce toxic seeds. Some species are known to cause dermatitis. The spurges, of which there are a great many in the United States, are toxic to hogs under some conditions. The spurge nettle *Jatropha stimulosa* is a powerfully irritating substance and causes dermatitis.

Fagropyrism

The European writers like Glasser and Pfeiler mention poisoning from buckwheat and smart weeds. Most of the trouble comes from feeding the hulls several cases of buckwheat hull poisoning have been reported to me. The symptoms are crying, twitching of the extremeties, the animal loses sense of touch. The several smart weeds produce a dermatitis in hogs.

Frosted Rape

Dr. C. H. Banks of Tipton reports that while frost is still on rape, toxin of some kind will cause a sloughing of the udder in swine. Further reports on the subject are desirable. Frosted alfalfa is said also to be injurious to swine.

Cottonseed Meal

Much has been written on poisoning from cottonseed meal. It causes suppressed appetite, weakness, unsteady gait and, in severe cases, blindness and inflammation of the digestive organs.

Squirrel Tail Grass—Awned Brome Grass

These two grasses are common in northern United States. They are a not uncommon cause of injury to swine through a mechanical action of the barbs. Of course strictly speaking this is not a poisoning. Ninety per cent of the pastures of Iowa con-

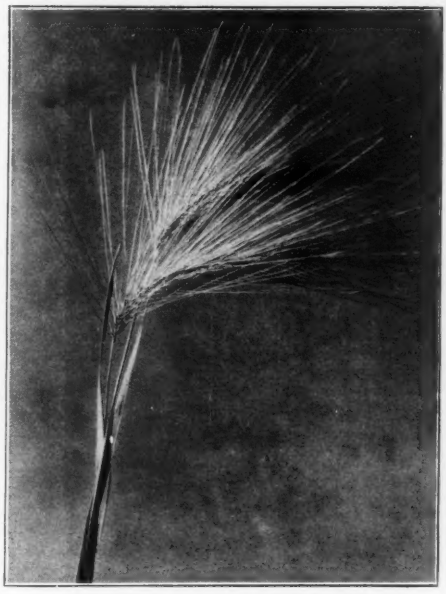
tain squirrel tail grass. The long awns are more or less purplish in color. The head breaks up in pieces and this is where the danger lies. The awned brome grass resembles cheat or chess but the chaffy scales have a bent awn. This grass is more common westward, as in Utah, Colorado and Nebraska. It is becoming common in Western Iowa. The awns of the squirrel tail grass, when taken into the mouth, break up into numerous sections, scatter within the mouth, adhere to the mucus membranes and cause inflammation. The action of the brome grass is very similar.

Nutritional Diseases

A great deal of trouble is caused by want of proper dietary which may be in a general way classified under the subject of poisons in the broadest sense. Many papers prepared by E. B. Hart and McCollum[16] and more recent papers by E. B. Hart and H. Steenbock[17] give excellent evidence to show that proper growth and reproduction of swine are closely related to the proper rations. Thus, for instance, whole grains fed as a sole article of the dietary and not supplemented with common salt or salt of natural water cause a nutritional failure. These authors state: "In some cases the first litter produced was apparently normal but imperfect nutrition was cumulative in its effect and increasing numbers of dead pigs at birth resulted in the second and third litters."

In a paper by E. B. Hart, W. S. Miller and E. V. McCollum[18] a statement is made that toxicity manifests its action by producing important histological changes in the nervous system of the animal not unlike those recorded for beri-beri. From their investigation it also appears that toxic conditions may arise in the hog, although apparently there is no toxic materials in the plant, owing to the absence of a proper salt mixture.

In the bulletins of the Wisconsin Agricultural Experiment Station, H. L. Russell and F. B. Morrison[19] in reviewing some of the work of Hart and Steenbock state: "Young growing pigs raised on a ration of yellowcorn, oil meal, floats, common salt, showed distinct nutritional disturbances after feeding for three months on the ration. This disturbance was easily corrected by the administration of cod liver oil at the rate of 10 cc per day." The above diseases have come under a general name of ricketts in swine. Apparently it is a question here of proper nutrition.



Hordeum jubateum (squirrel tail)

Another very important contribution due to malnutrition is a paper by L. A. Maynard, S. A. Goldberg and R. C. Miller[20] in a study of the dietary relationships and the pathology of stiffness in swine. This paper has a fine bibliography.

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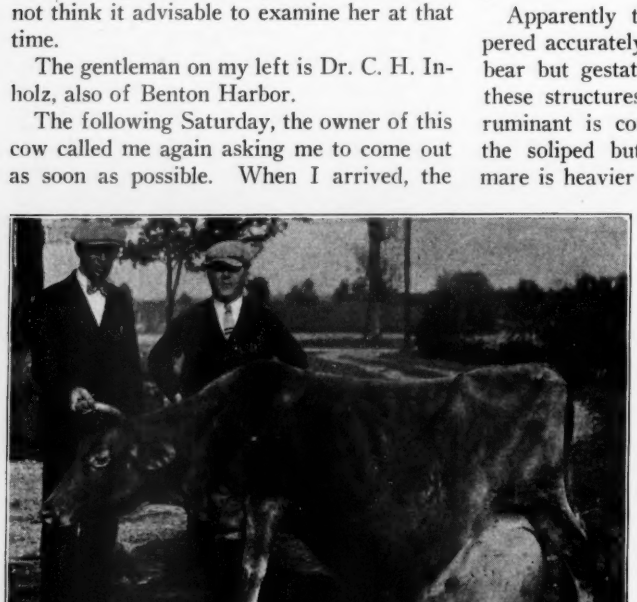
[1] Das kranke Schwein.
[2] Die Krankheiten des Schweines.
[3] Lehrbuch der Schweinezucht.
[4] Swine Diseases.
[5] Kansas State Agricultural College Circular.
[6] Bull. U. S. Dept. Agr. 1274, 1924.
[7] North American Veterinarian, 9:33, July, 1928.
[8] North American Veterinarian, 9:25-26, June, 1928.
[9] American Veterinary Medical Association 66:91-93.
[10] Veterinary Medicine, 23: 361, No. 8, August, 1928.
[11] Science N. S. 61:341.
[12] Bull. Fla. Agric. Exp. Station 196:337.
[13] Bull. N. Y. Agric. Ex. Sta. 144.
[14] Bull. Col. Agric. Exp. Sta. 316:13.
[15] Bull. Nev. Agric. Exp. Sta. 109.
[16] Journal Biol. Chem. 19:373.
[17] Journal Biol. Chem. 39:209.
[18] Jour. Biol. Chem. 25:239.
[19] No. 352.
[20] Bull. Cornell Univ. Agric. Exp. Sta. Memoir 86.

RUPTURE OF PREPUBIAN TENDON IN A COW

The accompanying illustration is of a case I had in June, 1928. I was called to see this cow, a Jersey, ten years old, on Tuesday. The owner said she had been in this condition for seven weeks. I was called because he thought she was due to calve. Since she was not in labor I advised him to let her go another day or two, as I did not think it advisable to examine her at that time.

The gentleman on my left is Dr. C. H. Inholz, also of Benton Harbor.

The following Saturday, the owner of this cow called me again asking me to come out as soon as possible. When I arrived, the cow had already calved with no trouble. It was a fine bull calf, and mother and calf did well.



Ventral Hernia due to rupture of the prepubian tendon.

The owner turned the cow and calf into pasture near the barn and in two weeks' time, her udder became very sore from dragging on the ground, and he decided to have her killed. I had intended to get a photograph of the cow and calf, but did not get to do so before she was destroyed.

I have seen several cases of ruptured prepubian ligaments, but never one such as this with the cow in normal condition, so far as feeding and getting around was concerned. It did not seem to bother her in the least.

Benton Harbor, Mich. J. M. Miller.

Comment by Prof. W. L. Williams: Rupture of the prepubian tendon, as this appears to be, is very rare in the cow. I recall but one case in my personal experience.

Apparently the abdominal floor is tempered accurately to the visceral load it is to bear but gestation places a new load upon these structures. The visceral load of the ruminant is comparatively greater than in the soliped but the gravid uterus of the mare is heavier than that of the cow, hence the percentage increase in the weight of the abdominal contents during pregnancy is much greater in the mare than in the cow. The pregnant cow is recumbent far more than the mare and thus relieves the strain. I have also he'd, in my Veterinary Obstetrics, that there is probably a definite disease and degeneration of the prepubian tendon in the mare.

When the abdominal floor of adult cattle gives way sufficiently to permit the rumen to gain entrance into the rupture, the hernia becomes enormous, and since the cow is comparatively short limbed, the descent of the hernial sac becomes peculiarly marked

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Hyperdermosis of goats has been reported in Teheran. As many as 30 larvae occur in a single goat. The larvae are about one-half inch in length. The adult parasite has not been identified. This would appear to be another condition that should be given consideration in goat importations into the United States.

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YOUR DOG AND MINE*

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Control of Rabies—The Japanese Method

Because of the great difficulty of enforcing dog-muzzling regulations, except where such regulations are supported by an aroused and enlightened public sentiment, such as occurs only locally and in the presence of an outbreak of the disease, some other method of control of rabies has been earnestly sought by veterinary authorities. As a result of this endeavor a vaccine for the prevention of rabies in dogs was developed and given an extensive trial in Japan five or six years ago.

More than 100,000 dogs in Tokio and Yokohama were given this vaccination. Of these only 41 contracted rabies. A showing that becomes even more remarkable when it was learned upon investigation that a majority of these 41 dogs were already infected by rabies when the vaccine was given, that is, they had been bitten by a rabid animal when vaccinated but had not yet developed the disease. The vaccine does not protect an infected dog. It must be given before the dog is exposed to rabies, that is, before it is bitten by a rabid animal.

The investigation of this vaccine was taken up immediately by biologic houses in America. This vaccine has now been given to more than a million dogs in this country, but no adequate records have been kept; and as there has been some hesitancy to accept the Japanese records, the value of the vaccine is still questioned.

A number of cities in the United States have adopted the vaccine method for the control of rabies, requiring all dogs to be vaccinated before they are licensed and prosecuting vigorously the extermination of stray, ownerless and unlicensed dogs. There has been some opposition to it, just as there was opposition in the beginning to the use of diphtheria antitoxin, and to railroads and electric street cars, and all other innovations.

One disadvantage of the Japanese method is the necessity of repeating the vaccination. The immunity following vaccination is not permanent. The vaccination needs to be repeated every year. Another disadvantage is the cost. Veterinarians usually charge from $2.00 to $5.00 for the treatment. Ordinarily, the lesser amount in cities where vaccination is compulsory. In some instances as in Tulsa, Oklahoma, the license fee was reduced to offset the cost of vaccination. It costs no more now in Tulsa to license a dog and to give him this protection against rabies than it formerly cost to license him without this protection. Another disadvantage of the vaccination is an occasional failure to give protection against the disease. This, however, is a weakness common to all preventive and curative treatments for all diseases.

Dog owners have in the main welcomed the opportunity to protect their pets and indirectly themselves and others by this means. The concern of owners for their dogs is always very great in the face of an outbreak of rabies, and any measure that offers them protection is earnestly sought. By the old method of controlling rabies, a person offers his own dog no protection by muzzling it, and this has proved the greatest weakness of the measure. Under the Japanese method, the owner by having his dog vaccinated protects it from rabies whether others have their dogs vaccinated or not.

* The articles under this head are part of a series of fifty-two discussions written by the editor of VETERINARY MEDICINE and distributed by a newspaper syndicate organization to various daily newspapers for use in their Saturday editions each week. Although written exclusively for lay readers they are offered here, with permission of the syndicate, for such suggestions as they may contain, for the veterinarian who desires to instruct his clients in the care of their dogs, with a view to increasing the popularity of this animal among those wsom he serves. Where some repetition is noted, it should be remembered that the articles were published separately at weekly intervals and it was necessary that each be in a measure complete.

As a public health measure, the successful vaccination of dogs for the prevention of rabies is of the greatest importance. As an economic measure it will save the loss of thousands of valuable food producing animals, a much larger number of dogs, and the cost of the Pasteur treatment for perhaps 50,000 persons yearly in this country alone.

As a humane measure, successful vaccination for the prevention of rabies, will without harm or discomfort, prevent indescribable suffering in many thousands of animals yearly. It only remains for it to be clearly and convincingly demonstrated that the vaccination is really a success in the prevention of rabies for the method to be widely adopted. Some cases of paralysis in dogs have developed vaccination by the Japanese method. That they were caused by the vaccine is probable but not certain. In any case the percentage has been but a fraction of 1%, and most of the cases have recovered.

NOTE: Since the foregoing was written (August, 1926) the number of dogs that have been given antirabic vaccine has increased very largely, but unfortunately the rceords are not such as to justify definite conclusions as to its protective value. In cities where large numbers of dogs have been vaccinated and large numbers left unvaccinated, the incidence of rabies in the latter has averaged perhaps 30 times the incidence in vaccinated dogs. In some cities the results have been far more favorable to the vaccination than this. A factor that needs to be taken into consideration, however, is the fact that under the conditions prevailing in cities where a large amount of vaccination of dogs for the prevention of rabies has been done, the vaccinated dogs have been well-cared-for dogs and the unvaccinated dogs in large part strays. Rabies is notoriously more prevalent among strays than among cared for dogs; just how much more prevalent, no one knows. The general belief among veterinarians is that vaccination of dogs for the prevention of rabies is a highly effective means of controlling the disease, but is not an infallible preventive of the disease.

Vaccination Against Rabies in Detroit

The examination of dogs' brains at the laboratories of the health department of Detroit resulted in finding 30 cases of rabies in 1921; 19 cases in 1922; 24 cases in 1923; 199 cases in 1924, and 80 cases in 1925. It is not probable that this represents all the cases of rabies occurring among dogs in Detroit, for the five-year period, but it probably reflects the relative prevalence of the disease for those years and is a good illustration of the usual occurrence of rabies, a disease of dogs that is prone to run along for years at what might be termed a low level and then to flare up suddenly without observable cause and increase in prevalence many fold.

Sometimes these outbreaks occur over a wide area as was the case in Iowa in 1925 when more than one-half of the state experienced a great prevalence of rabies; or the outbreak may be limited to a city as it was in Detroit in 1924, and Louisville, Kentucky, in 1926. Always following an outbreak of rabies in a district public opinion is aroused and the officials responsible for the enforcement of muzzling ordinances, quarantine regulations and other measures for the control of the disease are spurred to greater activity, and as a result of these measures the prevalence of the disease subsides after a time to the old level; the public forgets it; the vigilance of the regulatory officers relaxes and the stage is set to enact the same cycle over again.

Five cases of rabies were found by the Detroit health department laboratories in the first five months of 1923, and 21 times that number for the same period of time in 1924. This great prevalence had the usual result of stimulating efforts aimed at the control of the disease. An ordinance was passed classing all unlicensed dogs as strays, and requiring all licensed dogs permitted on the streets to be held

on a leash, muzzled or vaccinated against rabies. That is, if an owner wished to give his dog liberty on the streets he could lawfully do so only by muzzling it or having it given anti-rabies vaccination.

During the fiscal year that this ordinance went into effect, there were 12,000 dogs vaccinated, 20,000 licensed dogs unvaccinated, and 10,000 (estimated) strays. During the period 171 unvaccinated dogs developed rabies and 6 vaccinated dogs developed the disease. Of these 6, however, two developed the disease within a few days and cannot be counted as a failure of the vaccination which is ineffective during the incubation stage of the disease.

If we add these two to the cases in unvaccinated dogs, since they were unvaccinated when infected, we have 30,000 unvaccinated dogs furnishing 173 cases of rabies and 12,000 vaccinated dogs furnishing four cases of the disease. An incidence of the disease of 5.8 per thousand for the unvaccinated and of .33 for the vaccinated. To put it another way, rabies was more than 18 as frequent among the unvaccinated as among the vaccinated dogs. In the following fiscal year, there were no cases of rabies among the 4,000 dogs vaccinated that year and 87 cases of rabies among the unvaccinated, which includes 35,000 licensed and 50,000 (estimated) unlicensed dogs.

Health Commissioner Vaughn expresses himself as preferring the Detroit ordinance to one making no requirements as to vaccination and states that there have been no untoward results to the dogs following vaccination.

Detroit's experience with vaccination for the prevention of rabies supports the plan but is inconclusive. In the first place, four cases of rabies among 12,000 vaccinated dogs is a higher percentage than we should expect judging by the experience of Tokio and Yokohama with a very much larger number of vaccinated dogs, and again there were too many stray dogs in Detroit, particularly in 1925. As is recognized by all, rabies is far more prevalent among stray dogs than among dogs cared for by their owners, in fact this class (strays) is the chief problem in the control of rabies.

It has been pointed out by some opposed to the vaccination of dogs, that Detroit with vaccination has had less success in bringing its rabies outbreak under control and has been slower in getting the number of cases back to what might be termed normal than other communities where vaccination was not employed. While this is true, it is obviously not because of the vaccination, but in spite of it. The large number of strays in 1925, more than half of the canine population no doubt in the main, accounts for the persistence of the epizootic in Detroit. If the provisions of the ordinance were carried out, unlicensed dogs killed, and licensed dogs confined, muzzled or vaccinated, there probably would not have been a single case of rabies in Detroit in the fiscal year ending August 1, 1926.

NOTE—Twelve of this series of popular articles on "Your Dog and Mine" were reprinted in VETERINARY MEDICINE during the summer and fall of 1927. There have been numerous requests that more of the series be published and this will be done from time to time. Quite a number of subscribers have asked for separate reprints of the articles published, suitable for enclosures with letters, invoices and statements or other mail sent to patrons and to hand to dog owners when they call at the office. Any others desiring reprints of the former articles, or of these, may procure them made up in four-page pamphlets of a size suitable for enclosing, without folding, in a No. 6¾ envelope. The cost is merely nominal and depends largely upon the number ordered at a time; in quantities of 1,000 or more running about one-half cent each. The articles published in 1927 may be found in the August, September, October and November issues; in 1928 articles of this series have appeared in the August, September and November issues.

REMOVAL OF SALIVARY CALCULI

When treating a 12-year-old gelding for some minor thigh injury, the owner of the animal drew my attention to a sausage-like enlargement in the left cheek in the region of the buccinator muscle, ca. 4 inches long and 1½ inch in diameter. It appeared to be slightly moveable and painless. On examination under artificial light the tubercle at the end of Steno's duct in the vicinity of the third upper molar tooth as well as the opening of the duct itself appeared considerably enlarged. A small probe carefully inserted into the duct encountered resistance by striking a hard substance. Food-consumption and mastication was normal. The owner was informed that the enlargement in question might be due to the presence of salivary

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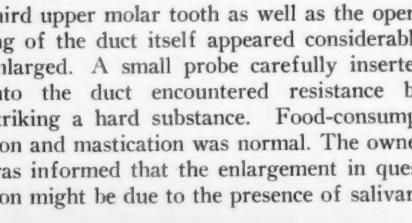

Twelve salivary calculi removed from a 12-year-old gelding. The operation was performed from the buccal cavity and was followed by an uneventful recovery. The largest calculus weighed 6¼ drams, the smallest weighed ¼ dram.

calculi, or probably of some other foreign substance, in the parotid duct and that surgical removal was indicated.

Since long distance and other circumstances did not permit the patient to be sent to the hospital, old-time methods were resorted to and, after having enjoyed a draught with two ounces of chloral hydrate thoroughly, he was gently placed upon "terra firma." The opening of Steno's duct in the buccal cavity was enlarged with a long fistula knife sufficiently to allow the introduction of an equally long forceps in order to grasp the foreign body. Rather with difficulty, a salivary calculus was eventually extracted which was of cylindrical shape, ca. 1 inch in diameter, of white color, with somewhat rough surface on its periphery and on end facing the opening of the duct, whilst its opposite end was quite smooth. Further examination revealed the presence of other calculi. Every effort to remove same with forceps failed. By elevating the head and manipulating the whole enlargement with the left hand from the buccal cavity, with the right one from the outside, eleven stones could be sqeezed out, the last one of the size of a small bean, uneven, with three very smooth surfaces. No further calculi could be detected. After the duct had been irrigated with Flavisol solution the patient was released. Food was entirely withheld for the first two days, the horse having access to good drinkwater all the time. Pilocarpin was administered hypodermically in order to have smaller stones which might be possibly elsewhere in the duct, removed with increased salivation.

On the ninth day after the operation, the wound in the buccal cavity seemed very well healed up. Today, two months after operation, the left cheek is completely normal, the sausage-like enlargement having disappeared completely.

Some of the large stones were crushed and consisted of a white, very hard, homogenous mass, the center appearing somewhat porous, but without a central nucleus. The smaller stones showed very smooth surfaces irregular in form and number where friction had taken place, the surface in contact with the duct being rough.

C. H. Grunert,
Fort Saskatchewan, Alberta, Can.

Regardless of the fact that the best flavored meats are obtained from mature animals there is an increasing demand for baby beef and lambs. The fattening and marketing of young animals permits of a quick turnover and by many feeders is claimed to be more profitable than the feeding and marketing of mature animals. It is probable, therefore, that it is the producer and not the consumer that is stimulating the demand for meats from young animals.